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Part II

Environmental Protection Agency

40 CFR Part 112

Oil Pollution Prevention; Non-Transportation Related Onshore Facilities; Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 112

[FRL–5909–5]

Oil Pollution Prevention; Non-Transportation Related Onshore Facilities

AGENCY: Environmental Protection Agency (EPA).

ACTION: Denial of petition requesting amendment of the Facility Response Plan rule.

SUMMARY: EPA is denying the request submitted by various trade associations to amend the Facility Response Plan (FRP) rule that the Agency promulgated under section 311(j) of the Clean Water Act (CWA), as amended by the Oil Pollution Act (OPA) of 1990. These organizations had requested that EPA modify the FRP rule in a number of ways to treat facilities that handle, store, or transport animal fats and vegetable oils in a manner differently from those facilities that store petroleum-based oils. EPA believes that the petition did not substantiate the claimed differences between animal fats and vegetable oils and petroleum oils so as to support a further differentiation between these groups of oils under the FRP rule. Instead, EPA continues to find that a worst case discharge or substantial threat of discharge of animal fats and/or vegetable oils to navigable waters, adjoining shorelines, or the exclusive economic zone could reasonably be expected to cause substantial harm to the environment, including wildlife that may be killed by the discharge of fats or vegetable oils. Moreover, EPA believes that in setting different response strategies for petroleum and non-petroleum oils, (with animal fat and vegetable oils in the latter category), the FRP rule already provides for adequate differentiation in response planning requirements for all covered facilities.

ADDRESSES: The official record for this decision is located in the Superfund Docket, at the U.S. Environmental Protection Agency, [Docket Number SPCC–3]. The docket is available for inspection between 9 a.m. and 4 p.m., Monday through Friday, excluding Federal holidays, at US EPA Crystal Gateway 1 (CG1), 1235 Jefferson Davis Highway, Arlington, VA 22202. Appointments to review the docket can be made by calling 703–603–8917. The public may copy a maximum of 266 pages from any regulatory docket at no cost. If the number of pages copied exceeds 266, however, a charge of 15 cents will be incurred for each additional page, plus a $25.00 administrative fee.

FOR FURTHER INFORMATION CONTACT: Bobbie Lively-Diebold, Oil Pollution Center, Office of Emergency and Remedial Response (5203G), U.S. Environmental Protection Agency, 401 M Street, SW., Washington, DC 20460 at 703–356–8774 (lively.barbara@epamail.epa.gov); or the RCRA/Superfund Hotline at 800–424–9346 (in the Washington, DC metropolitan area, 703–412–9810). The Telecommunications Device for the Deaf (TDD) Hotline number is 800–553–7672 (in the Washington, DC metropolitan area, 703–412–3323).

SUPPLEMENTARY INFORMATION: The contents of this Denial of Petition are listed in the following outline:

I. Background

The OPA (Pub. L. 101–380, 104 Stat. 484) was enacted to expand prevention and preparedness activities, improve response capabilities, ensure that shippers and oil companies pay the costs of spills that do occur, provide an additional economic incentive to prevent spills through increased penalties and enhanced enforcement, establish an expanded research and development program, and establish a new Oil Spill Liability Trust Fund administered by the U.S. Coast Guard.

Section 4202(a) of the OPA amends CWA section 311(j) to require regulations for owners or operators of facilities to prepare and submit ''a plan for responding, to the maximum extent practicable, to a worst case discharge, and to a substantial threat of such a discharge, of oil or a hazardous substance.'' This requirement applies to all offshore facilities and any onshore facility that, ''because of its location, could reasonably be expected to cause substantial harm to the environment by discharging into or on the navigable

waters, adjoining shorelines, or the exclusive economic zone'' (''substantial harm facilities'').

On July 1, 1994, EPA published its Final Rule amending the Oil Pollution Prevention regulation (40 CFR part 112) to incorporate new requirements to implement amended section 311(j)(5) of the CWA. (Oil Pollution Prevention; Non-Transportation-Related Onshore Facilities; Final Rule, 59 FR 34070, July 1, 1994). Under authority of section 311(j)(1)(C) of the CWA, the Final Rule also requires planning for a small and medium discharge of oil, as appropriate.

In the final rule, EPA determined that for the purposes of section 311(j) planning, the OPA includes non-petroleum oils. The Agency noted that the definition of ''oil'' in the Clean Water Act includes oil of any kind, and that EPA uses this broad definition in 40 CFR part 110, Discharge of Oil. Animal fats and vegetable oils fall within the CWA definition of ''oil.''

Only a small number, no more than 1¼ percent of the total SPCC community regulated (approximately 5,400 of a total of 435,000 facilities) under 40 CFR part 112.1–112.7 meet the criteria for substantial harm under 40 CFR 112.20. Only a small number of the 5,400 substantial harm facilities (an estimated 50 to 100) store or use vegetable oil and animal fat and have prepared and submitted FRPs.

A. The Organizations' Petition

By a letter dated August 12, 1994, EPA received a ''Petition for Reconsideration and Stay of Effective Date'' of the OPA-mandated FRP final rule as that rule applies to facilities that handle, store, or transport animal fats or vegetable oils. The petition was submitted on behalf of seven agricultural organizations (''the Organizations'' or ''Petitioners''): the American Soybean Association, the Corn Refiners Association, the National Corn Growers Association, the Institute of Shortening & Edible Oils, the National Cotton Council, the National Cottonseed Products Association, and the National Oilseed Processors Association.

To support the Petition, the Organizations referenced an industry-sponsored report titled ''Environmental Effects of Release of Animal Fats and Vegetable Oils to Waterways'' (prepared by ENVIRON Corporation, June 28, 1993), and an associated study titled ''Diesel Fuel, Beef Tallow, RBD Soybean Oil and Crude Soybean Oil: Acute Effects on the Fathead Minnow, Pimephales Promelas'' (prepared by Aqua Survey, Inc., May 21, 1993). Both the report and the study had been submitted to EPA during the facility response plan rulemaking as enclosures to a comment filed over nine months after the close of the comment period. Based, in part, on these studies (the ENVIRON report and Aqua Survey study), the Petitioners asked EPA to create a regulatory regime for response planning for non-petroleum, ''non-toxic'' oils separate from the regime established for petroleum oils and ''toxic,'' non-petroleum oils.

The report and the study provided information on certain physical, toxicological, and chemical properties of animal fats and vegetable oils compared with other types of oil. The petitioners argued that according to the ENVIRON report, the presence of animal fats and vegetable oils in the environment does not cause significant harm. Six specific conclusions of the ENVIRON report regarding vegetable oils and animal fats were that these substances are not toxic to the environment; are essential components to human and wildlife diets; readily biodegrade; are not persistent in the environment like petroleum oils; do have a high Biochemical Oxygen Demand (BOD), which could result in oxygen deprivation where there is a large spill in a confined body of water that has low flow and dilution; and can coat aquatic biota and foul wildlife.

The Petitioners also submitted an Appendix to their Petition that included specific suggested language to amend the July 1, 1994, FRP rule. The submitted language would have resulted in the following changes regarding facilities that handle, store, or transport animal fats and vegetable oils: Further clarified the definition of animal fats and vegetable oil (set out in Appendix E, 1.2 of the FRP); allowed mechanical dispersal and ''no action'' options to be considered in lieu of the oil containment and recovery devices otherwise specified for response for a worst case discharge; required the use of a containment boom only for the protection of fish and wildlife and sensitive environments; and increased required on-scene arrival time for response resources from 12 hours (including travel time) to 24 hours plus travel time for medium discharges and worst case Tier 1 response resources.

The Federal natural resource trustee agencies, including the Fish and Wildlife Service (FWS), had reviewed the ENVIRON study. In an April 11, 1994, letter to the Department of Transportation's (DOT) Research and Special Projects Administration (RSPA), the FWS stated that the Report did not provide an accurate assessment of the dangers that non-petroleum oils pose to fish and wildlife and environmentally sensitive areas. The letter stated that the key facts were misrepresented, incomplete, or omitted in the Report. FWS also observed that the ENVIRON report failed to give appropriate significance to the fouling potential of edible oils (USDOI/FWS, 1994).

The National Oceanic and Atmospheric Administration (NOAA) also had evaluated the effects on the environment of spilled non-petroleum oils, including coconut, corn, cottonseed, fish, and palm oils. (Memorandum of Record, dated June 3, 1993, from the Department of Commerce (DOC)/NOAA Hazardous Materials Response and Assessment Division.) The NOAA assessment, based on literature research, addresses physical and chemical properties and toxicity of these and other oils, and indicates that some edible oils, when spilled, may have adverse environmental effects. (The views of the FWS and NOAA on the adverse effects of animal fats and vegetables are discussed in detail in the preamble to the U.S. Coast Guard's final rule setting forth response plan requirements for marine transportation-related facilities, [61 FR 7890, 7907–7908, Feb. 29, 1996] and are included in the docket that supports this decision. These views also are discussed in EPA's Request for Data and Comment on Response Strategies for Facilities That Handle, Store, or Transport Certain Non-Petroleum Oils, 59 FR 53742–53743, October 26, 1994.)

On October 26, 1994, in view of the differing scientific conclusions reached by the Petitioners, the FWS, and other groups and agencies, EPA requested broader public comment on issues raised by the Petitioners in a notice and request for data (Request for Data and Comment on Response Strategies for Facilities That Handle, Store, or Transport Certain Non-Petroleum Oils, 59 FR 53742, October 26, 1994). These issues included whether to have different specific response approaches for releases of animal fats and vegetable oils (rather than increased flexibility), and the effects on the environment of releases of these oils. EPA also asked commenters to recommend specific data that relate to the comparison of petroleum and non-petroleum oils. EPA received fourteen comments in response to its October 26, 1994, notice and request for data.

Of these fourteen commenters, most agreed with the trade associations' request that EPA should modify the FRP rule. Most of the commenters asserted that, based upon the ENVIRON report, animal fats and vegetable oils are readily biodegradable and not persistent

in the environment. Certain commenters also argued that vegetable oils and animal fats are less toxic than other types of oils. Other commenters argued that edible oils pose less risk to the environment because they are typically stored in smaller tanks at food processing facilities, whereas petroleum-based oils are stored in larger tanks at petroleum facilities. One commenter, citing the unnecessary and burdensome regulations and the excellent spill record of the animal fat and vegetable oil industry, stated that EPA should differentiate animal fats and vegetable oils from other types of oils. One commenter questioned the accuracy of the ENVIRON report and stated that non-petroleum oils can adversely affect fish and wildlife and environmentally sensitive areas.

B. Background on the Processing and Storage of Vegetable Oils and Animal Fats

In 1992, approximately 20.8 billion pounds of vegetable oils and animal fats were consumed in the United States, including over 14.8 billion pounds for edible uses; and more than 5.9 billion pounds for inedible uses, such as soap, paint or varnish, feed, resins and plastics, lubricants, fatty acids, and other products (Hui, 1996a). The extent of processing of vegetable oils and animal fats depends on the ultimate use of the product. Chemical composition, which determines the toxicity and fate of oils in the environment, changes at each step in processing, as impurities or specific components are removed or chemicals formed; chemical composition can also be changed by storage, heating, or reactions in the environment (Hui, 1996d; Brekke, 1980).

Processing steps in vegetable oil facilities are generally independent operations that are not connected by continuous flow, and between each processing step there may be one or more storage tanks (Hui, 1996d). Many crude vegetable oil storage tanks, which are usually constructed of welded carbon steel, have a capacity of 1 million pounds (approximately 140,000 gallons) (Hui, 1996d). They may be located in the open or enclosed in a structure. Storage tanks for finished fats and oils are generally made of iron, stainless steel, or aluminum and typically hold between 75 and 200 tons (about 21,000 to 56,000 gallons) of product.

In a typical integrated vegetable oil processing facility, steps may include crude oil storage, preparation, extraction and meal finishing, removal of gums and lecithin processing, caustic refining, bleaching and dry removal of gums and waxes, hydrogenation, interesterification, fractionation, deodorizing, and shortening or margarine production (Hui, 1996d; Brekke, 1980). During these steps, several classes of materials may be removed, such as gums, phospholipids, pigments, free fatty acids, color bodies, pigments, metallic prooxidants, and residual soaps. New compounds, including oxidation products, polymers and their decomposition products, may be formed and contaminants introduced during processing (Hui, 1996d).

Impurities are also removed and chemical structure modified during processing of animal fats (Hui, 1996d). The major animal fats are lard and tallow. Steps in the processing of animal fats may include rendering, bleaching, hydrogenation, deodorizing, interesterification, and fractionation. Rendering, the removal of fat from animal tissues using heat or mechanical means, is often a continuous process that results in products that require no further treatment. Further refining removes materials, such as free fatty acids or collagen or protein, or changes the characteristics of the fat for specialized use.

Spills of crude vegetable oils containing gums, phospholipids, free fatty acids, and a host of other chemical components can differ greatly from spills of processed oils in their persistence in the environment, the environmental compartments in which they are distributed, the breakdown products that they form, their rate of degradation, and the exposure and environmental effects that they produce. Some minor components of oils can affect their properties or cause adverse health and environmental effects. Spilled oils and fats can be transformed by physical, chemical, or biological processes to form products that are more or less toxic than the original oil, depending on the specific oil and the products that are formed.

The EPA has considered the Petitioners' claims in detail. EPA's technical evaluation on the Petitioners' claims is set forth in section II. EPA's responses to suggested changes in the FRP regulation are provided in section III. Detailed studies and information to support this document are provided in a Technical Document, which is located in the Docket.

II. Technical Evaluation of Petitioners' Claims

A. General

The Petitioners claim that unlike most if not all other oils, animal fats and vegetable oils are non-toxic, readily biodegradable, not persistent in the environment, and in fact are essential components of human and wildlife diets. Most of the Petitioners' arguments focus on toxicity, although toxicity is only one of several mechanisms by which oil spills cause environmental damage.

In making its claims, the Petitioners have disregarded fundamental scientific principles and ignored a large body of scientific evidence that was considered by EPA in its promulgation of rules implementing the requirements of the CWA. The ENVIRON report submitted by the Petitioners acknowledges that animal fats and vegetable oils can cause oxygen deprivation and coating of animals, but the Petitioners incorrectly minimize the importance of these mechanisms in causing environmental damage and rely instead on limited studies in narrow areas of toxicity, which are then improperly generalized to support the Petitioners' claims.

Petitioners' submission emphasizes that animal fats and vegetable oils are used by all organisms for food. The ingestion of small quantities of edible oils by humans, however, is a completely different situation from spills of oil into the environment. These situations differ markedly in the extent and duration of exposure, the route of exposure, the species exposed, the composition of the chemicals involved, the circumstances surrounding the exposure, and the types of effects produced—factors that determine the toxicity and severity of the adverse effects of chemicals. Thus, even if the human consumption of small quantities of oils in food were judged completely safe, no inferences could be drawn about the toxicity and other effects of vegetable oils and animal fats on environmental organisms exposed in the very different circumstances of oil spills.

The Petitioners' arguments about toxicity do not address the central issue: Spills of animal fats and vegetable oils kill or injure fish, birds, mammals, and other species and produce a host of other undesirable effects. Whether this death and destruction results from toxicity or from other processes, spills of animal fats and vegetable oils should be prevented and if spills occur, quickly removed to reduce the environmental harm and other adverse effects they produce.

B. Petitioners' Claim: Animal Fats and Vegetable Oils Are Non-Toxic

The Petitioners claim that EPA's implementation of the response plan provisions and other regulatory changes

under the CWA are inconsistent with established regulatory principles and with the available scientific data related to animal fats and vegetable oils, which, unlike other oils, are non-toxic.

EPA Response: For a number of reasons that are detailed in this document and the Technical Document, EPA disagrees with the Petitioners' contention that animal fats and vegetable oils are non-toxic when spilled into the environment. First, while the Petitioners rely on laboratory tests that measure only the acute lethal effects of some vegetable oils and animal fats in one species of fish, these tests say nothing about other acute toxic effects or long-term toxic effects, or toxic effects on other species or ecosystems, or toxic effects of oil spilled in the environment under conditions that differ from those in the laboratory. Second, the tests submitted by the Petitioners cannot demonstrate ''non-toxicity'' of vegetable oils and animal fats; indeed the tests described in the study only measure the lethality of the oils tested under a given set of experimental conditions. Third, other information and data indicate that animal fats and vegetable oils, their components, and degradation products are not as ''non-toxic'' as the Petitioners assert. Fourth, while low levels of certain animal fats and vegetable oils or their components may be essential constituents of the diet of humans and wildlife, adverse effects occur from exposure to high levels of these chemicals. Numerous examples in the scientific literature demonstrate that essentiality does not confer safety and essential elements can produce toxic effects (Klaassen et al., 1986; NAS, 1977a; Rand and Petrocelli, 1985; Hui, 1996b).

Furthermore, EPA emphasizes that toxicity is only one of several mechanisms by which oil spills cause environmental damage. As discussed below, the physical effects of spilled oil—such as coating animals and plants with oil and suffocation of aquatic organisms from oxygen depletion—and the destruction of the food supply kill birds and mammals, destroy fish and other aquatic species, and damage their habitats.

By contaminating food sources, reducing breeding animals and plants that provide future food, contaminating nesting habitats, and reducing reproductive success through contamination and reduced hatchability of eggs, even oils that remain in the environment for relatively short periods of time can cause long-term deleterious effects years after the oil was spilled.

1. How Animal Fats and Vegetable Oils Produce Adverse Environmental Effects

The deleterious environmental effects of spills of petroleum oils and non-petroleum oils, including animal fats and vegetable oils, are produced through physical contact and destruction of food sources as well as toxic contamination (USDOC/NOAA, 1996; NAS, 1985e; Crump-Wiesner and Jennings, 1975; Frink, 1994; Frink and Miller, 1995; Hartung, 1995; USDOI/FWS, 1994). Nearly all of the most immediate and devastating environmental effects from oil spills—such as smothering of fish or coating of birds and mammals and their food with oil—are physical effects related to the physical properties of oils and their physical interactions with living systems (Hartung, 1995).

While these immediate physical effects and effects on food sources may not be considered the result of ''toxicity'' in the classic sense—i.e., effects that are produced when a chemical reacts with a specific receptor site of an organism at a high enough concentration for a sufficient length of time (Rand and Petrocelli, 1985)—severe debilitation and death of fish and wildlife are caused by spills of animal fats and vegetable oils, other non-petroleum oils, and petroleum oils and their products. Adverse environmental effects can occur long after the initial exposure to animal fats and vegetable oils because of toxicity, persistence of products in the environment, or destruction of food sources and habitat and diminished reproduction resulting from physical effects or toxicity.

2. Physical Properties

Petroleum oils and non-petroleum oils, including vegetable oils and animal fats, share common physical properties and produce similar environmental effects (Crump-Wiesner and Jennings, 1975; USDOI, 1994; Frink, 1994). When spilled in the aquatic environment, petroleum oils, animal fats and vegetable oils and their fatty acid constituents may float on the water's surface, become solubilized or emulsified in the water column, or settle on the bottom as a sludge, depending on their physical and chemical properties (Crump-Wiesner and Jennings, 1975; DOC/NOAA, 1992, 1996). Vegetable oils and animal fats that are solid at room temperature still serve as potent physical contaminants and are much more difficult to remove from affected animals than petroleum oil (Frink, 1994).

While the physical properties of vegetable oils and animal fats are highly variable, most fall within in a range that is similar to the physical parameters for petroleum oils. (See Appendix I, Table 1: Comparison of Physical Properties of Vegetable Oils and Animal Fats With Petroleum Oils and Table 2: Comparison of Vegetable Oils and Animal Fats with Petroleum Oils). Common properties—such as solubility, specific gravity, and viscosity—are responsible for the similar environmental effects of petroleum and vegetable oils and animal fats. Petroleum and vegetable oils and animal fats can enter all parts of an aquatic system and adjacent shoreline, and similar methods of containment, removal and cleanup are used to reduce the harm created by spills of petroleum and vegetable oils and animal fats.

3. Chemical Composition

The chemical composition and physical properties of petroleum and non-petroleum oils, including vegetable oils and animal fats, determine their fate in the environment (where they go, reactions, rate of disappearance) and the exposure and adverse effects that they produce. The chemical composition changes at each step in processing, as impurities or specific components are removed or chemicals formed (Hui, 1996d; Brekke, 1980). Chemical composition can also change with storage, heating, or reactions in the environment.

The main constituents of vegetable oils and animal fats are esters of glycerol and fatty acids (Hui, 1996b). The ester linkages can be hydrolyzed to yield free fatty acids and glycerol. While triglycerides (triacylglycerols) predominate, fats and oils also contain mono- and diglycerides (mono-and diacylglycerols) and other lipids, e.g., phosphatides and cholesterol, free fatty acids, and small amounts of other compounds. Fats and oils also contain other minor components, such as polynuclear aromatic hydrocarbons (PAHs). Like vegetable oils and animal fats, petroleum crude oils are hydrocarbon mixtures that can be further processed to make specific products; but the hydrocarbon constituents of petroleum oils are primarily alkanes (paraffins), cycloalkanes, and aromatic hydrocarbons (IARC, 1989).

Fatty acids largely determine the chemical and physical properties of triglycerides (Hui, 1996a) and influence their fate and effects in the environment. The structure of the fatty acids can change as they are processed, stored, heated, or transformed by physical, chemical, and biological processes in the environment. The fatty acid composition of vegetable oils and

animal fats varies with plant or animal species, season, geographical location, feed, and other factors.

The physical and chemical properties of petroleum and non-petroleum oils can change after they have spilled into the environment. Spilled oil can be transformed through a wide variety of physical, chemical, and biological processes (USDOC/NOAA, 1992a, 1996). These processes are affected by many factors, among them temperature, oxygen, light, ionizing radiation, and the presence of metals (Kiritsakis, 1990; Hui, 1996a, 1996d).

As the composition of the oil changes, so does its fate in the environment and its toxicity. The products that are formed can be more or less toxic than the original oil, depending on the specific oil and the products that are formed. Oxidation of vegetable oils and animal fats, which may contribute rancid off-flavors and odors, can create products, such as cyclic monomers and oxycholesterols that are toxic at relatively low concentrations (Hui, 1996a). Polymers of soybean oil and sunflower oil can form concrete-like aggregates with soil or sand that cannot be readily degraded by bacteria and remain in the environment for many years after they are spilled (Minnesota, 1963; Mudge, 1995, 1997a, 1997b). Petroleum oils also undergo oxidation and polymerization reactions and can form tars that persist in the environment for years (NAS, 1985d).

4. Environmental Effects

Spills of petroleum and vegetable oils and animal fats can harm aquatic organisms and wildlife in many ways (Crump-Wiesner and Jennings, 1975):

- Oil can coat the feathers and fur of birds and mammals and cause drowning and hypothermia and increased vulnerability to starvation and predators from lack of mobility.
- Oils can act on the epithelial surfaces of fish, accumulate on gills, and prevent respiration. The oil coating of surface waters can interfere with natural processes of reaeration and photosynthesis. Organisms and algae coated with oil may settle to the bottom with suspended solids along with other oily substances that can destroy benthic organisms and interfere with spawning areas.
- Oils can increase BOD and deplete water of oxygen sufficiently to kill fish.
- Oils can cause starvation of fish and wildlife by coating food and removing the food supply. Animals that ingest large amounts of oil through contaminated food or preening themselves may die as the result of the oil ingested. Animals can also starve because of increased energy demands needed to maintain body temperature when they are coated with oil.
- Oils can exert a direct toxic action on fish, wildlife, or their food supply.
- Oils can taint the flavor and cause intestinal lesions from laxative properties in fish.
- Oils can foul shorelines and beaches. Oil spills can also create rancid odors.

The environmental effects of vegetable oils and animal fats and petroleum oils, their chemical and physical properties, and their environmental fate are compared in Appendix I, Table 2.

a. Physical Effects of Spilled Oil. Physical effects produce nearly all of the most immediate and devastating environmental effects from oil spills. Even oils that remain in the environment for relatively short periods of time can cause long-term deleterious effects years after the oil was spilled.

Coating with Oil. Among the immediate effects of oil spills is the coating of the feathers of birds and fur of mammals (Hartung, 1995). Coating of animals and their food supply is produced by spills of petroleum and non-petroleum oils alike. Birds and some mammals, such as sea otters and river otters that depend upon entrained air for buoyancy and insulation, are particularly vulnerable to harm from spills of non-petroleum and petroleum oils (NAS, 1985e; Hartung, 1967, 1995). In freshwater or tidal brackish waters, oiled birds are usually waterfowl and wading birds, such as herons (Alexander, 1983).

Birds and mammals become coated with oil when they land in an oil slick or surface from underneath (Hartung, 1995). Oil alters the structure and function of the feathers and fur by disrupting their orderly arrangement, thereby reducing entrainment of air and causing loss of buoyancy and thermal insulation (Rozemeijer, 1992; Leighton, 1995; Frink and Miller, 1995; NAS, 1985e; Alexander, 1983; Hartung, 1967, 1995; Crump-Wiesner and Jennings, 1975). As the plumage absorbs water, the weight and body mass of the birds increases, and the birds sink and may drown. Birds and mammals, with feathers or fur matted down by petroleum or non-petroleum oils, can also die from hypothermia and/or dehydration and diarrhea or fall victim to predators.

Birds that are able to endure excess chilling while avoiding their predators may reach shore and sit or stand in a state of shock (NAS, 1985e; Alexander, 1983). To maintain body temperature, such birds would have to eat twice the normal amount of food; yet they are often isolated from their food supply (Hartung, 1967, 1995; Alexander, 1983). Fat and muscular energy reserves of these birds are rapidly exhausted and their body temperature drops (Hartung, 1967; Croxall, 1977; Alexander, 1983; Rozemeijer et al., 1992). As their appetite declines, death from starvation ensues. Similarly, sea otters with fur coated with oil require increased metabolism to compensate for major changes in conductance and heat flow across the body surface (Hartung, 1967, 1995; Kooyman, 1977; Williams et al., 1990; NAS, 1985e).

Oiled birds tend to preen their feathers and may ingest large amounts of oil from attempting to clean themselves and from consuming oil-contaminated food and oil particles (Frink, 1994; Frink and Miller, 1995; Alexander, 1983; NAS, 1985e; Hartung, 1965, 1967, 1995). Bird rescuers have described dead birds with organs filled with oil from eating oiled food (Lyall, 1996; Frink and Miller, 1995). Oil can also be transferred to birds through consumption of fouled prey or direct contact with the oiled shoreline or surface water (Frink and Miller, 1995; Smith and Herunter, 1989). The coated birds that are observed after oil spills are probably a small proportion of the total affected, as weakened birds are likely victims of predators (Hartung, 1995; Alexander, 1983; NAS, 1985e; Lyall, 1996; Frink and Miller, 1995; McKelvey et al., 1980; Smith and Herunter, 1989; Minnesota, 1963).

Small spills of vegetable oil, animal fat and petroleum oils can cause great ecological damage, depending upon the location of the spill and other factors. Even a small spill of vegetable oil can be far more damaging to aquatic birds than certain petroleum oils (McKelvey et al., 1980; Smith and Herunter, 1989).

Suffocation. Suffocation and death of fish and other biota are often the consequence of oxygen depletion of the water. Oxygen depletion can result from reduced oxygen exchange across the air-water surface below the spilled oil or from the high BOD produced by microor ganisms degrading oil (Crump-Wiesner and Jennings, 1975; Mudge, 1995). While a higher BOD is associated with greater biodegradability, it also reflects the increased likelihood of oxygen depletion and potential suffocation of aquatic organisms under certain environmental conditions (Crump-Wiesner and Jennings, 1975). Oxygen depletion and suffocation are produced by petroleum and non-petroleum oils, including animal fats and vegetable oils. Under certain conditions, however, some vegetable oils and animal fats

present a far greater risk to aquatic organisms than other oils spilled in the environment, as indicated by their greater BOD.

According to studies designed to measure the degradation of fats in wastewater, some food oils exhibit nearly twice the BOD of fuel oil and several times the BOD of other petroleum-based oils (Groenewold, 1982; Institute, 1985; Crump-Wiesner and Jennings, 1975). While the higher BOD of food oils is associated with greater biodegradability by microorganisms using oxygen, it also reflects the increased likelihood of oxygen depletion and suffocation of aquatic organisms under certain environmental conditions (Groenewold, 1982; Institute, 1985; Crump-Wiesner and Jennings, 1975). Oil creates the greatest demand on the dissolved oxygen concentration in smaller water bodies, depending on the extent of mixing (Crump-Wiesner and Jennings, 1975).

Contamination of Eggs. After spills of non-petroleum and petroleum oils, oil can be transferred from birds' plumage to the eggs they are hatching. Petroleum and non-petroleum oils, including vegetable oils and animal fats, can smother an avian embryo by disrupting the egg/air interface, sealing pores, and preventing gas exchange (Albers, 1977; Szaro and Albers, 1977; Leighton, 1995; USDOI, 1994).

In addition to the severe physical effects produced by non-petroleum and petroleum oils, some petroleum oils can also damage embryos apparently through mechanisms of toxicity (Albers, 1977; Szaro and Albers, 1977; Leighton, 1995; Szaro, 1977; NAS, 1985e). Very small quantities of petroleum or crude oil cause mortality and developmental effects in avian embryos from a wide variety of species (Leighton, 1995; NAS, 1985c). Whether vegetable oils and animal fats can harm embryos through toxicity as well as physical effects is unknown, for no studies of the toxicity of vegetable oils and animal fats to avian embryos and developing birds were located.

b. Effects of Oil on Metabolic Requirements. To survive spills of petroleum and non-petroleum oils, animals require increased energy (NAS, 1985e; Hartung, 1967, 1995). Birds coated with oil must eat twice their food ration to maintain body temperature (Hartung 1967, 1995). Yet birds are often isolated from their food sources following an oil spill or find their food coated with oil (Hartung 1967, 1995). Sublethal effects can increase vulnerability to disease or decrease growth and reproductive success, although the individual may continue to live for some time (NAS, 1985e; Frink and Miller, 1995; Smith and Herunter, 1989).

Studies of polluted animals show that physiological stress is manifested in higher energy demand (Sanders et. al., 1980). When increasing environmental stress greatly elevates metabolism and reduces assimilation, little energy remains for growth and reproduction, so that most species disappear and only a few tolerant species survive in chronically polluted environments. Oil pollution also forces animals to turn from the most economical biochemical pathways to other more costly physiological pathways.

c. Effects of Oil on Food and the Food Web, Communities, and Ecosystems. The effects of oil on the food web and community structures depend on the type and amount of oil spilled, the physical nature of the area, nutritional status, oxygen concentration, and previous exposure of the impacted area (NAS, 1985e). Geographic location appears far more important in determining the impacts of oil spills than spill size (Frink and Miller, 1995; McKelvey et al., 1980). The community structure and activities of microbes that degrade petroleum oil are affected by both catastrophic and chronic spills. The risks from oil spills can be shifted from those associated with toxicity to those associated with habitat, e.g., predator-prey interaction (NAS, 1985e).

The vulnerability of species and individuals to oil spills varies greatly (NAS, 1985e), and the extent and rate of recovery depends on many factors. In enclosed waters where recruitment of organisms from outside becomes less important, intrinsic factors may limit the recovery of the zooplankton community. Plant communities too can be affected long after an oil spill, with imbalances persisting for a decade or more, even after the floral community is reestablished (Sanders et al., 1980). When diversity and density have increased and stabilized many years after a spill, behavioral responses may continue to be distorted or biochemical pathways may be shifted from efficient to more costly pathways.

d. Indirect Effects. While not generally regarded as classic ''toxicity,'' high levels of fatty acids and triglycerides from vegetable oils and animal fats can upset the fermentation and digestion of ruminants, such as cattle, goats, deer, antelope, sheep, moose, buffalos, and bighorn sheep (Van Soest, 1994). Although intake of normal levels of lipids does not affect fermentation in ruminants, excess unsaturated fatty acids and triglycerides can profoundly suppress essential fermentation bacteria and alter fermentation balance, lipid metabolism, and milk fat production. Methane suppression is likely with a single large dose of unsaturated oil that exceeds the threshold of tolerance by fermentation bacteria. A practical limit for fat of about 8–10% of dietary dry matter is expected (personal communication, D. Ullrey, 1996).

Indirect effects also occur when petroleum oil is spilled in the environment (NAS, 1985e). After a spill of number 5 fuel oil, the herring population was reduced because of increased fungal damage to fish eggs, which in turn resulted from a decreased population of amphipods which graze fungi growing on fish eggs.

5. Toxicity

Adverse effects occur through both non-toxic and toxic mechanisms. Whether an adverse effect occurs through toxicity or other mechanisms is often unknown (Yannai, 1980). For example, birds exposed to spilled oil may die from non-toxic mechanisms —starvation, hypothermia, drowning, shock, susceptibility to predators because of a food supply that is inadequate to support increased energy requirements, and consumption of oiled food or oil from preening that clogs their organs— or from the toxicity of chemicals or biotransformation products in the oil. The deaths of the birds occur, regardless of the mechanisms involved or knowledge about these mechanisms.

Toxicology is the study of the adverse effects of chemicals on living organisms, including lethality; reproductive effects; effects on development; cancer; effects on the nervous system, kidney, liver, immune system, or other organs; and biochemical effects, such as enzyme inhibition (Klaassen et. al., 1986; Rand and Petrocelli, 1985). To examine the nature of toxic effects and evaluate the probability of their occurrence, factors that affect toxicity must be known. A brief discussion of toxicity is presented below. The supporting Technical Document discusses toxicology in greater depth.

a. Principles of Toxicology. The toxicity of chemicals depends on factors that are related to the organism itself, chemical composition, external environmental factors, and the exposure situation. The necessity of considering many factors in the evaluation of toxicity is underscored in basic textbooks about toxicology, such as *Casarett and Doull's Toxicology* that state:

''* * * Whether or not a toxic response occurs is dependent * * * on the chemical

and physical properties of the agent, the exposure situation, and the susceptibility of the biologic system or subject. Thus to characterize fully the potential hazard of a specific chemical agent, we need to know not only what type of effect it produces and the dose required to produce the effect but also information about the agent, the exposure, and the subject * * *'' (Amdur *et al.,* 1991).

The hazards and risks from environmental exposures to chemicals are assessed with toxicological studies in the laboratory and with epidemiological studies, while field studies may be used to assess the ecological effects of chemicals on multiple species or ecosystems (NAS, 1985c; NAS, 1977a; OSTP, 1985; Rand and Petrocelli, 1985). Toxic chemicals enter the body primarily by ingestion, inhalation, and skin contact (Klaassen et al., 1986). The toxic effects from acute exposure to a chemical (e.g., a single dose during a short period of time such as 24 hours) may differ greatly from those produced by long-term (chronic) exposures. Toxic effects can be immediate or they can be delayed.

A substance that is harmless at low concentrations in food may be hazardous if it comprises a large portion of the diet. Because there is little margin of safety for many of the elements to which people are exposed daily, the daily intake of many elements in the diet, such as iron, could not be increased 5 or 10 times without adverse effects (Klaassen *et al.,* 1986).

b. Exposure From Oil Spills. Spills of petroleum and vegetable oils and animal fats during processing, storage, and transportation can result in acute or chronic exposures to fish and wildlife. Not only massive spills but small quantities that are spilled repeatedly may result in environmental harm (Alexander, 1983; McKelvey et al., 1980; Smith and Herunter, 1989). Small volume spills can produce severe environmental damage because of the behavior of oils in the environment, their physical effects, and the toxicity of some oil constituents and transformation products. Many of the immediate, devastating effects of spilled petroleum and vegetable oils and animal fats, such as coating, suffocation, and other physical effects, occur during acute exposures. Long-term effects have also been reported from spills of petroleum oil, vegetable oils and animal fat.

During an oil spill, the potential for significant exposures is very high (Hartung, 1995). Unlike laboratory experiments using controlled amounts of oil, large amounts of oil may be released during spills. While the initial mortalities of birds and mammals exposed to spilled oil are usually from drowning or hypothermia resulting from coating, the ingestion of oil begins to contribute to effects later as birds consume large amounts of oil through preening or ingestion of oil-contaminated food and oil particles (Hartung, 1967, 1995). Fish and other aquatic organisms may die from suffocation soon after an oil spill or exhibit toxic effects, including cancer and adverse effects on growth and reproduction, following acute or chronic exposures to spilled oils and fats or their breakdown products.

Spilled oil can be transformed through a wide variety of physical, chemical, and biological weathering processes that change oil composition, behavior, exposure routes, and toxicity (USDOC/NOAA 1992, 1996). Whether the environmental fate and toxicity of the transformation products differs from that of the parent depends upon the specific oil and products that are formed.

c. Toxicity of Petroleum Oils. The toxic effects of petroleum oils are summarized in Appendix I, Table 2. The effects of petroleum oils have been investigated extensively in many species (NAS, 1985e; IARC, 1984; Albers, 1995). Commonly reported individual effects of petroleum oils include impaired reproduction and reduced growth as well as death in plants, fish, birds, invertebrates, reptiles and amphibians; blood, liver, and kidney disorders in fish, birds, and mammals; malformations in fish and birds; altered respiration or heart rate in invertebrates, fish, reptiles, and amphibians; altered endocrine function in fish and birds; altered behavior in many animal species; hypothermia in birds and mammals; impaired salt gland function in birds, reptiles, and amphibians; altered photosynthesis in plants; and increased cells in gills and fin erosion in fish. Among the group effects of petroleum are changes in local population and community structure in plants, invertebrates and birds and changes in biomass of plants and invertebrates.

Petroleum oils affect nearly all aspects of physiology and metabolism and produce impacts on numerous organ systems of plants and animals as well as altering local populations, community structure, and biomass (Albers, 1995; NAS, 1985e). Impaired reproduction, reduced growth and development, malformations, behavioral effects, blood and liver and kidney disorders, altered endocrine function, and a host of other effects of petroleum oils on organisms have been reported.

Certain petroleum products and crude oil fractions are associated with increased cancer in refinery workers and laboratory animals (IARC, 1989). Many of these petroleum oils contain benzene and polynuclear aromatic hydrocarbons (PAHs), toxic constituents that are carcinogenic in humans and animals. Untreated and mildly treated mineral oils are carcinogenic to humans. In experimental animals, some distillates and cracked residues derived from the refining of crude oil and residual (heavy) fuel oils are carcinogenic. There is limited evidence in experimental animals for the carcinogenicity of unleaded automotive gasoline, fuel oil number 2, crude oil, and naphtha and kerosene produced by certain processes.

d. Toxicity of Vegetable Oils and Animal Fats. The toxicity of vegetable oils and animal fats and the toxic effects on many systems and organs in the body are summarized in Appendix I, Table 2 and described briefly below. A detailed discussion of these effects is included in the supporting Technical Document.

The acute and chronic toxicity of vegetable oils and animal fats, types of fats, and their components and degradation products have been evaluated in toxicology and epidemiological studies. Chemical and physical properties of the particular animal fat or vegetable oil, the exposure situation, the biologic systems exposed, and the environmental conditions that are present are factors that influence the toxicity of a chemical.

Acute lethality tests are among several measures used to evaluate acute toxicity. They can be employed to rank chemicals or to screen doses that may be selected for longer term toxicity testing, or they can be an early step in tiered hazard assessment approaches. The use of different protocols and test species in acute lethality tests makes comparisons between tests difficult. For example, although the Petitioners claim that the tests conducted by Aqua indicate that smaller amounts of petroleum oils than certain vegetable oils and animal fats kill half the population of some aquatic species; other acute lethality studies suggest that by one measure, vegetable oils are more toxic than petroleum-derived mineral oil. In studies comparing the acute lethality of corn oil, cottonseed oil, and petroleum-derived mineral oil in albino rats, no rats receiving mineral oil died, while smaller doses of the vegetable oils administered for a shorter time period killed rats (Boyd, 1973).

Vegetable oils and animal fats produce other types of acute toxicity as well. Like petroleum oils, vegetable oils

and animal fats are laxatives that can produce diarrhea or cause lipid pneumonia in animals. These effects can compromise the ability of animals in the wild to escape their predators (USDOI, 1994; Frink, 1994). Clinical signs of toxicity in rats fed large amounts of corn oil or cottonseed oil for 4 or 5 days include decreased appetite, loss of body weight, abnormal lack of thirst, diarrhea, fur soiling, listlessness, pale skin, incoordination, cyanosis (dark blue skin color from deficient oxygenation of the blood), and prostration, followed by respiratory failure and central nervous system depression, hypothermic coma, and death. Autopsies of the rats showed violent local irritation of the gastrointestinal tract, which allowed the absorption of oil droplets into the bloodstream and deposition of oil in tissues, resulting in inflammation, congestion in the blood vessels, dehydration, degenerative changes in the kidney, loss of organ weights, and stress reaction (Boyd, 1973).

Animals exposed to vegetable oils and animal fats can manifest a range of chronic toxic effects. High levels of some types of fats increase growth and obesity but cause early death in several species of animals and may decrease their reproductive ability or the survival of offspring (NAS/NRC, 1995). On the other hand, the growth of some fish decreases with elevated levels of vegetable oils (Salgado, 1995; Mudge 1995, 1997a). Mortality of mussels exposed to one of four vegetable oils began after 2 or 3 weeks of exposure. Growth inhibition, effects on shells and shell lining, and decreases in foot extension activity that are essential to survival were observed in mussels exposed to low levels of sunflower oil.

Dietary fat consumption has been associated with the incidence of some types of cancer, including mammary and colon cancer, in laboratory animals and humans (Hui, 1996a; USDHHS, 1990; FAO/WHO, 1994). The intake of dietary fat or certain types of fat has also been correlated with the incidence of coronary artery disease, diabetes, and obesity in epidemiological studies (Hui, 1996a; FAO/WHO, 1994; Nelson, 1990; Katin at al, 1995). High dietary fat intake has also been linked to reduced longevity and altered reproduction in laboratory animals and altered immunity, altered steroid excretion, and effects on bone modeling and remodeling in humans.

Some vegetable oils and animal fats contain toxic constituents, including specific fatty acids and oxidation products formed by processing, heating, storage, or reactions in the environment (Hui, 1996a; Berardi and Goldblatt, 1980; Yannai, 1980; Mattson, 1973). Toxic effects on the heart, red blood cells, and immune system; effects on metabolism; and impairment of reproduction and growth can be caused by constituents or transformation products of vegetable oils and animal fats. In addition, some constituents of vegetable oils and animal fats cause cancer in rainbow trout, while lipid oxidation products may play a role in the development of cancer and atherosclerosis (Hendricks at al 1980a and 1980b).

Acute Toxicity: Acute Lethality Test (LC_{50} Test) Submitted by Petitioners. The tests by Aqua that were submitted by the Petitioners are acute lethality tests that measure only the death of organisms. These tests provide no data on nonlethal acute toxicity, including irreversible damage, or long-term effects experienced by organisms and ecosystems. The LC_{50} (lethal concentration 50) value or LD_{50} (lethal dose 50) value does not describe a ''safe'' level but rather a level at which 50% of test organisms are killed under the experimental conditions of the test (Rand and Petrocelli, 1985; Klaassen *et al.*, 1986). (A high LC_{50} value indicates low acute lethal toxicity, for a large concentration of chemical is needed to cause 50% mortality.) If the Aqua test results were accurate, they would indicate that diesel fuel kills half the population of fathead minnows at lower concentrations than aerated crude soybean oil, RBD soybean oil, and beef tallow. Spills of petroleum oils, vegetable oils and animal fats that result in LC_{50} concentrations in the environment could kill half the organisms with sensitivity similar to fathead minnows when conditions are identical to those in the Aqua tests.

Although the manner in which the Aqua tests were conducted precludes accurate determination of the LC_{50} values, the tests nevertheless demonstrate that petroleum oils and vegetable oils and animal fats can injure and kill fish by toxicity or oxygen depletion and suffocation. In the first set of the Aqua tests, all of the minnows exposed to diesel fuel and unaerated crude soybean oil died. The fish surfaced and gulped for air or swam spasmodically before dying, just as they do in the environment when suffocating from oxygen depletion following spills of petroleum and non-petroleum oils, including vegetable oils and animal fats.

Results Questionable. However, the test procedures used by Aqua render questionable the results suggesting that diesel fuel is more deadly at lower concentrations than soybean oil. The procedures deviate in important ways from standardized methodology, although the Aqua report states that test procedures are based on accepted methodologies. Appendix I, Table 3: Comparison of Aqua Methods and Standard Acute Aquatic Testing Methods lists key differences between the methods used by Aqua and the standard methods referenced in the Aqua report as well as more recent methods published by these same organizations that were omitted from the Aqua report. The accuracy of the LC_{50} estimates provided by Aqua is highly doubtful because of the following deficiencies:

- Oxygen depletion. In the first set of Aqua tests, dissolved oxygen was below acceptable levels in the vessels with crude soybean oil. It is impossible to determine whether oxygen depletion or toxicity killed fish.
- Short exposure period. The Aqua tests were conducted for only 48 hours, instead of the 96 hours used in most methods. Fish that are alive at 48 hours may not survive for 96 hours.
- Unknown concentrations of test material encountered by fish during the test: (1) Oil sheens floated on test solutions and cloudiness was so severe that fish could not be observed for 24 hours; (2) the Aqua report contained no data on actual chemical concentrations of parent chemical or breakdown product, a critical determination in static tests where concentrations change over time (Rand and Petrocelli, 1985; NAS, 1985c). Aqua relied instead on the original nominally designated concentrations that are highly dubious, especially given the turbidity of the test solutions that cleared up over the course of the test, the likely degradation of test material in the aerated test system, and the use of vessels that were not stainless steel or glass and may have adsorbed test material; (3) the Aqua test did not aerate all test solutions and controls, did not maintain dissolved oxygen concentration at 80% or more of the nominal concentration, and did not test non-aerated and aerated oils together—requirements of standardized methods that allow gentle aeration. If vegetable oils degrade rapidly, as Petitioners claim elsewhere, aeration will increase the degradation of the oils in the test system; (4) the Aqua report provided no data on oil particle size, even when visual inspection showed that solutions of test material were cloudy and the NAS study referenced in the report cautioned against relying on visual inspections of clarity (NAS, 1985c); and (5) improper data reporting and evaluation. Results from two dissimilar tests were combined, although the tests

lacked a common test substance, used different test conditions, failed to measure actual concentrations, and included no estimates of variability between the two sets of tests. Aqua also failed to provide data on confidence intervals and slopes, as required by all of the standardized methods referenced by Aqua and by the Aqua protocol.

Relevance of Acute Lethality Tests to Spills in the Environment Challenged. Serious questions remain about the relevance of the LC_{50} laboratory results to spills in the environment (NAS, 1985c, 1985e). The many test variables that influence estimates of LC_{50}—including the nature of the chemicals or mixtures tested, test parameters (e.g., route and method of administration, frequency and duration of exposure, mixing energy, temperature, salinity, static vs. flow-through systems, duration of observations) and biological factors (e.g., species selected for testing, sex, age or life-stage, weight, contamination history of the organism)—rarely reflect the conditions that occur following a spill (Rand and Petrocelli, 1985; NAS, 1985c; Wolfe, 1986; Abel, 1996). The water-soluble fraction used in static tests does not simulate the dynamic process of the change in stages between aqueous and oil phases that depends on parameters unique to each spill (NAS, 1985c). Once oil is spilled in the environment, the composition, concentration, and toxicity of oil and its components can be profoundly altered by chemical and biological processes, such as evaporation and biological oxidation.

Further, acute lethality tests by their very nature usually provide no data on toxic effects other than death (NAS, 1985c; Rand and Petrocelli, 1985; Klaassen *et al.,* 1986). Indeed, a widely-used toxicology text warns that ''defining acute toxicity based only on the numeric value of an LD_{50} is dangerous'' (Hayes, 1982). Animals that survive a toxic response nevertheless may suffer irreversible damage (NAS, 1985e). These nonlethal, adverse effects must be considered in assessing the risks of chemical exposure. Nor do acute lethality tests measure long-term effects or effects on ecological communities or changes in predator-prey relationships which occur, for example, when animals coated with spilled oil are weakened and become more susceptible to predators.

Acute Toxicity: Other Acute Lethality Tests (Aquatic Tests). (See Appendix I, Table 2, for other aquatic lethality information.) Free fatty acids are among the products formed from vegetable oils and animal fats by processing, storage, heating, or reactions in the environment. Static tests with juvenile fathead minnows indicate that oleic acid, which is found in Canola, safflower, and sunflower oils, is more acutely lethal at 96 hours than at 24 hours and is intermediate in lethality in tests of a series of 26 organic compounds (USEPA, 1976; Hui, 1996a).

Acute Toxicity: Other Acute Lethality Tests (Tests with Laboratory Animals). (See Appendix I, Table 2.) Studies comparing the acute lethality of corn oil, cottonseed oil, and mineral oil in albino rats show that by one measure cottonseed oil and corn oil are more toxic than petroleum-derived mineral oil, although interpretation of the studies is complicated by differences in the experimental protocol (Boyd, 1973). No albino rats receiving mineral oil by gavage (tube into stomach) for 15 days died, while smaller doses of cottonseed oil and corn oil administered for a shorter time period killed rats.

The toxic effects differed significantly in rats receiving corn oil or cottonseed oil and those administered mineral oil (Boyd, 1973). Clinical signs of toxicity in rats receiving corn oil or cottonseed oil included anorexia (decreased appetite), loss of body weight, abnormal lack of thirst, decreased urination, diarrhea, fur soiling, listlessness, pallor (pale skin), incoordination, cyanosis (dark blue skin color from deficient oxygenation of the blood), and prostration (Boyd, 1973). Rats administered corn oil died after respiratory failure and hypothermic coma, while death followed central nervous system depression and coma in rats ingesting cottonseed oil. Autopsies showed violent local irritation of the gastrointestinal tract that allowed the absorption of oil droplets into the bloodstream. Oil droplets were deposited in many body organs with resultant inflammation, vascular congestion, degenerative changes in the kidney, and other effects. In contrast, no deaths occurred among rats administered mineral oil for 15 days and clinical signs differed in many respects from those observed in rats treated with corn or cottonseed oil.

Chronic Toxicity. Appendix I, Table 2 summarizes the chronic toxicity of vegetable oils and animal fats and petroleum oils. Cancer and adverse effects on growth, reproduction, development, and longevity as well as other toxic effects have been observed in several species following chronic or subchronic exposures to vegetable oils and animal fats or their constituents. (Subchronic exposures are longer than acute exposures, generally 1–3 months for rodents and longer than 4 days for aquatic species.)

Dietary fat and some classes of fats that are found in vegetable oils and animal fats have been associated with the increased incidence of some types of cancer, including mammary and colon cancer, in laboratory animals and humans (Hui, 1996a; USDHHS, 1990; FAO/WHO, 1994). The intake of dietary fat or of certain types of fat has also been correlated with the incidence of coronary artery disease, diabetes, and obesity in epidemiological studies. High dietary fat intake has also been linked to reduced longevity and altered reproduction in laboratory animals and altered immunity, altered steroid excretion, and effects on bone modeling and remodeling in humans.

In addition, some vegetable oils and animal fats contain toxic constituents or form toxic degradation products, including specific fatty acids and oxidation products, when they undergo processing, heating, storage, or reactions in the environment. The toxic effects of these chemicals are summarized briefly in Appendix I, Table 2 and described further in section II.5.d Toxicity of Specific Fatty Acids and Other Constituents of Vegetable Oils and Animal Fats. Among the toxic effects observed after exposure to these chemicals are cardiac toxicity, rupture of red blood cells, growth suppression, anemia, impaired reproduction, and adverse effects on the immune system and metabolism. In addition, the cyclopropene fatty acid constituents of cottonseed oil and some other vegetable oils cause liver cancer in rainbow trout and increase carcinogenesis of other chemicals, and some oxidation products may play a role in the development of colon cancer and atherosclerosis.

Cancer. Unlike petroleum oils that contain a large proportion of PAHs, including some PAHs that are animal and/or human carcinogens, vegetable oils and animal fats contain only small amounts of PAHs (Kiritsakis, 1991; IARC, 1984). Dietary fat intake and consumption of some classes of fats that are found in vegetable oils and animal fats have been implicated in the development of certain types of cancer—including cancer of the breast and colon and probably cancer of the prostate and pancreas—in studies of laboratory animals and in epidemiological studies (NAS/NRC, 1985c; Hui, 1996a; USDHHS, 1990; FAO/WHO, 1994). An expert panel organized by two United Nations organizations concluded that abundant data show that animals fed high-fat diets develop tumors of the mammary gland, intestine, skin, and pancreas more readily than animals fed low-fat diets, although caloric restriction can override

the effect (WHO/FAO, 1994). Animal studies also indicate correlations between total fat intake and liver cancer and between high-fat diets and certain types of chemically-induced or light-induced skin tumors. Studies describing the relationships between fat consumption and cancer in animals and humans have been summarized recently (Hui, 1996a).

Development of some types of cancer is influenced by the type of fat consumed. Breast cancer increased (shortened latency period for tumor appearance, promotion of growth, and increased mammary tumor incidence) in rodents receiving diets rich in the essential fatty acid linoleic acid (polyunsaturated fatty acid or PUFA of the n-6 family) compared to rodents consuming diets high in saturated fatty acids (Hui, 1996a). In contrast, fish oil containing different fatty acids (n-3 PUFA) inhibited mammary tumor development, probably by inhibiting the effects of linoleic acid. The incidence of colon cancer is strongly associated with diet, especially diets high in total fat and low in fiber content in laboratory animals and epidemiological studies (Hui 1996a; USDHHS, 1990). Some types of fat, such as dietary cholesterol and certain long-chain fatty acids, have been proposed as colon cancer promoters, while other types of fat (n-3 PUFA) may inhibit development of colon cancer (Hui, 1996a).

Non-Carcinogenic Toxic Effects. The non-carcinogenic toxic effects of vegetable oils and animal fats on aquatic organisms and laboratory animals are summarized in Appendix I, Table 2, briefly described below and are discussed in greater detail in the Technical Document.

Non-Carcinogenic Toxic Effects on Mussels. The detrimental environmental effects of sunflower oil have been investigated extensively in laboratory studies and in the field at the site of the 1991 wreck of the cargo tanker M.V. Kimya, where much of its 1500-tonne cargo of crude sunflower oil was spilled over a 6–9 month period (Mudge *et al.*, 1993, 1994, 1995; Mudge, 1995, 1997b; Salgado, 1992, 1995). Mussels died in the intertidal shores at sites near the wreck; in other areas where mussels survived, their lipid profiles revealed an altered fatty acid composition reflecting the fatty acids in sunflower oil (Mudge et al., 1995; Mudge, 1995, 1997a, 1997b; Salgado, 1992, 1995). Mobile species that left the spill area were replaced with other species, affecting diversity.

Sunflower oil, olive oil, rapeseed oil, and linseed oil produced several types of adverse effects in mussels at low exposure rates in the laboratory (Salgado, 1995; Mudge, 1995; Mudge, 1997a). These four vegetable oils killed mussels or reduced their growth rate as much as fivefold within 4 weeks, even at low exposure rates (1 part of oil in 1000 in a flow-through sea water system). Mussels exposed to sunflower oil were more likely to die. Exposure to sunflower oil created behavioral differences in the mussels, such as decreased foot extension activity and altered gaping patterns. Interference with foot extension activity that allows the mussels to form threads for attachment to the substratum can dislodge mussels and endanger their survival; removal of the oil reversed the effect (Salgado, 1995).

All four oils killed mussels in mortality studies in the laboratory; 10% mortality was observed in mussels exposed to sunflower oil, rapeseed oil, or olive oil for up to 4 weeks, while 70% or 80% mortality was reported when mussels were exposed to linseed oil (Salgado, 1995; Mudge, 1997b). No control mussels died. Mussels began dying the second week after exposure to linseed or sunflower oil, and later when exposed to rapeseed or olive oil. Death may have been caused by suffocation in mussels that refused to gape in the presence of the oil or by formation of a toxic metabolite. The death of mussels in aerated growth tanks where anoxia (lack of oxygen) was not the cause of death suggests that vegetable oils kill mussels through mechanisms of toxicity.

The shells of mussels exposed to the vegetable oils in the laboratory lacked the typical nacre lining, perhaps because of altered behavior in the presence of oil stressors (Salgado, 1995; Mudge, 1997a). The internal shell surfaces of mussels treated with vegetable oils were chalky in contrast to controls that exhibited an iridescent luster. Prolonged closure of the mussels in response to oil can cause anoxia and increase the acidity of the internal water with dissolution of the inner shell.

Sunflower oil from the wreck of the M.V. Kimya polymerized in water and on sediments and formed hard ''chewing gum balls'' that washed ashore over a wide area or sank, contaminating the sediments inhabited by benthic and intertidal communities near the spill (Mudge, 1995). Concrete-like aggregates of sand bound together with sunflower oil remain on the shore near the site of the M.V. Kimya spill almost six years later (Mudge, 1995, 1997a, 1997b; Mudge *et al.*, 1995). In laboratory experiments with saltmarsh sediments simulating a spill over a 35-day period, linseed oil percolated rapidly through the sediments but sunflower oil polymerized and formed an impermeable cap, reducing oxygen and water permeability (Mudge *et al.*, 1995; Mudge, 1997a). In the environment, oxygen reduction would eventually produce anoxia in sediments with the death and removal of benthic organisms, changes in species from a community that is aerobic to an anaerobic community, and erosion of the saltmarsh sediments (Mudge *et al.*, 1994, 1995).

Non-Carcinogenic Toxic Effects on Fish. Other studies have also shown that exposure to an excess of fat or fatty acids can be detrimental to fish, even though fish and other aquatic organisms require certain essential fatty acids for growth and survival. Poor growth and low feed efficiency were observed in rainbow trout fed 4% or more of certain polyunsaturated acids (Takeuchi and Watanabe, 1979). High levels of dietary fatty acids reduced growth in channel catfish; while saturated, monounsaturated, or PUFA from fish oil enhanced channel catfish growth (Stickney and Andrews, 1971, 1972). Some dietary fatty acids inhibited the growth of common carp, but saturated and monounsaturated acids and other classes of polyunsaturated fatty acids from fish oil enhanced carp growth (Murray *et al.*, 1977). More recent papers show the relatively efficient use of high levels of dietary lipid by warmwater and coldwater fishes, provided essential fatty acid requirements are met (NAS/NRC, 1981a, 1983). Increased lipid intake, however, has been associated with increased deposition of body fat.

Non-Carcinogenic Toxic Effects on Laboratory Animals. The chronic toxic effects of petroleum oils and vegetable oils and animal fats on laboratory animals are summarized in Appendix I, Table 2 and detailed in the accompanying Technical Document. High levels of dietary fat have been associated with shortened lifespan and altered reproduction in laboratory animals (NAS/NRC, 1995). While 5% dietary fat is recommended for most laboratory animals, growth usually increases significantly when animals are fed higher levels of fat. Apparently, this increased growth comes at a high cost, however, for longevity is often reduced and reproduction may be affected adversely in animals consuming high levels of fat.

The relationship between dietary fat intake and kidney diseases has been demonstrated in laboratory animals (Hui, 1996a). Rats, rabbits, and guinea pigs fed high cholesterol diets developed kidney damage. Diets containing 2% cholesterol increased the

incidence or severity of coronary atherosclerosis in rats exposed chronically to the cold (Sellers and Baker, 1960). Histological aberrations in the small intestine and nearby lymph nodes have also been reported in rats consuming high doses of fish oil concentrate in a subchronic toxicology study (Rabbani et al., 1997).

Increasing the consumption of some dietary lipid components, such as oleic acid and cholesterol, also increases the need for other fatty acids in rats (NAS/NRC, 1995). The ratios of PUFA and polyunsaturated to saturated fatty acids greatly influence tissue lipids and the formation of important compounds, such as prostaglandins. The type of fat can influence bone formation rates and fatty acid composition of cartilage in chicks (Hui, 1996a).

Toxicity of Specific Fatty Acids and Other Constituents of Vegetable Oils and Animal Fats. In addition to the adverse effects produced in humans and other animals by high fat diets or by consumption of certain classes of fats and oils, toxic effects can be produced by constituents of some animal fats and vegetable oils, including specific fatty acids and gossypol, and their transformation products (Hui, 1996a; Berardi, 1980; Yannai, 1980; Mattson, 1973). While plant breeding and processing can reduce the levels of some constituents in the final product, the constituents are present during the early stages of processing and storage of some vegetable oils and may enter the environment. Although the development of varieties of glandless, gossypol-free cottonseed and new varieties of rape seed with little erucic acid have reduced these two constituents in some oils, gossypol is found in crude oils and in oils derived from older cottonseed varieties with greater resistance to disease and insects and high amounts of erucic acid are contained in rapeseed oil used for the manufacture of lubricants and fatty acid derivatives (Hui, 1996a, 1996b). Toxic materials can be formed during normal processing procedures, heating, and storage or by reactions that occur when such materials are released in the environment. Spills of crude vegetable oils may differ greatly in their toxicity and other effects from spills of processed vegetable oils and animal fats. Figure 1: Toxicity and Adverse Effects of Components and Transformation Products of Vegetable Oils and Animal Fats illustrates the variety of toxic effects that may be caused by constituents and breakdown products of vegetable oils and animal fats. For example, small amounts of gossypol are lethal when they are ingested for prolonged periods despite the relatively high LD_{50} values obtained in acute toxicity tests; fat accumulated in heart muscle of weanling rats after a single day of consuming diets containing erucic acid; and cyclopropene acids, such as sterculic acid, are liver carcinogens in rainbow trout (Berardi, 1980; Mattson, 1973; Hendricks et al., 1984). Phytoestrogens, which occur naturally in some legumes and oils, including soybean, fennel, coffee, and anise oils, exhibit estrogen-like activity in reproductive organs of laboratory animals (Hui, 1996a; Sheehan, 1995; Levy et al., 1995).

When vegetable oils are spilled, air, moisture and heat in the environment can cause these oils to form various harmful oxidation products, which may be more toxic than the original product. Releases of used oil from restaurants or releases of oil during refining may already contain toxic oxidation products that may be further oxidized in the environment. Cholesterol oxidation products or COPs that are formed by autooxidation of cholesterol when it is exposed to air, heat, photooxidation, and oxidative agents have numerous biological activities and may play a role in the development of atherosclerosis (Hui, 1996a). Lipid oxidation products (LOPs) that can be formed when unsaturated fatty acids are oxidized upon exposure to oxygen, light, and inorganic and organic catalysts have been associated with colon cancer (Hui, 1996a; Hoffmann, 1989; Lawson, 1995).

FIGURE 1. TOXICITY AND ADVERSE EFFECTS OF COMPONENTS AND TRANSFORMATION PRODUCTS OF VEGETABLE OILS AND ANIMAL FATS

Component or transformation products	Type of oil	Effects
Gossypol [1,2,3]	Cottonseed oil	Cardiac irregularity in several species of animals, death from circulatory failure or rupture of red blood cells and decreased oxygen-carrying capacity in blood.
		Discolors egg yolks in laying hens by interacting with yolk iron; effect decreased by ferrous sulfate, increased by cyclopropene fatty acids in cottonseed oil.
		Crosslinks proteins in several species; reduces protein quality, uncouples respiratory-linked energy processes, reduces activity of respiratory enzymes and protein kinases and proteins involved in sterol, steroid, and fatty acid metabolism.
		High LD_{50} in acute tests for mice and swine, but small amounts are lethal when ingested for prolonged period.
		Death from pulmonary edema in subacute poisoning; wasting and lack of assimilation of food with chronic poisoning.
		Depressed appetite, loss of body weight, diarrhea, effects on red blood cells, heart and lung congestion, degenerative changes in liver and spleen, various pathological effects depending on species.
		Body weight depression, reduced sperm production and motility in male rats; loss of appetite, diarrhea, hair loss, anemia, hemorrhages in stomach and intestines, congestion in stomach, intestines, lungs, and kidneys of rats.
		Spastic paralysis of hind legs, degeneration of sciatic nerve, rapid pulse, cardiac effects in cats.
		Posterior incoordination, stupor, lethargy, weight loss, diarrhea, vomiting, loss of appetite, lung and heart congestion, hemorrhaging of liver, fibrosis of spleen and gallbladder in dogs.
		Stupor, lethargy, loss of appetite, spastic paralysis, decreased litter weights, congestion of large intestine, hemorrhaging in small intestines, lungs, brain, and legs in rabbits.

FIGURE 1. TOXICITY AND ADVERSE EFFECTS OF COMPONENTS AND TRANSFORMATION PRODUCTS OF VEGETABLE OILS AND ANIMAL FATS—Continued

Component or transformation products	Type of oil	Effects
		Weight loss, decreased appetite, leg weakness, reduced red blood cells, congestion, vacuoles in liver, enlarged gallbladder and pancreas, decreased egg size, decreased egg hatchability, discolored yolk in poultry.
		Thumps or labored breathing, weakness, emaciation, diarrhea, enzyme effects, hair discoloration, dilated heart, reduced hemoglobin, lipid in kidneys, widespread congestion of organs in swine.
		Erratic appetite, breathing difficulties, fatty degeneration of liver, decreased blood clotting, and death in young calves but no toxicity in older ruminants.
		No human toxicity in China, where gossypol used as male contraceptive, antifertility reversible.
Erucic Acid [2,4,5]	Rapeseed oil, mustardseed oil.	Adverse effects on heart in laboratory animals; inflammation of heart in rat , fat deposition until fat content of heart 3 to 4 times normal, fat droplets visible in heart followed by mononuclear cell infiltration and replacement of fat and droplets with fibrous tissue in muscle; weanling rats accumulated fat in heart muscle after only one day; fatty infiltration of heart absent with fully hydrogenated rapeseed oil, indicating effects from erucic acid; erucic acid in heart muscle in rats exposed long-term; changes in skeletal muscle in rats.
		Lipid accumulation in hearts of rats, hamsters, minipigs, squirrel monkeys and ducklings; fluid accumulation around heart and liver cirrhosis in ducklings.
		Enlarged spleen, increased cell permeability and destruction of red blood cells in guinea pigs (erucic and nervonic acids in rapeseed oil).
		Growth suppression in rats, pigs, chickens, turkeys, guinea pigs, hamsters, and ducklings fed rapeseed oil; suppressed body weight gain in rats fed fats plus erucic acid.
		Degenerative changes in liver and kidney, fewer and smaller offspring in rats fed high levels of rapeseed oil.
Cyclopropene Fatty Acids [2,3,4,6,7,8,9,10].	Cottonseed oil, kapok seed oil, cocoa butter.	Discolors egg whites, can be removed by hydrogenation; growth suppression in rats; reduced comb development in roosters.
		Impaired female reproduction in laboratory animals and hens; depressed egg production, reversible in hens; embryomortality in hens and rats; developmental abnormalities in rats, increased mortality in rat pups.
		Liver carcinogen in rainbow trout; increases carcinogenic effects of other chemicals; adverse effects on cholesterol and fatty acid metabolism in several species; aortic atherosclerosis in rabbits; liver damage in rabbits and rainbow trout.
Oxidation Products [2,4,11,12,13,14,15].	Many vegetable oils and animal fats.	Cholesterol Oxidation Products (COPs): Numerous biological activities include adverse effects on blood vessels, destruction of cells, mutagenicity, suppression of immune response, inhibition of certain metabolic mechanisms; may contribute to development of atherosclerosis.
		Lipid Oxidation Products (LOPs): Associated with colon cancer; lipid peroxides act as cancer promoters or cocarcinogens and form crosslinks between DNA and proteins; lipid peroxidation correlated with severity of atherosclerosis.
		Oxidative fatty acid fraction of products of thermal and oxidative changes from prolonged heating of fats and oils in laboratory studies (may not simulate commercial heat treatment); severe heart lesions, distended stomach, kidney damage, hemorrhage of liver and other tissues, reduced liver enzyme activity in laboratory animals; reduced body weight gain and feed consumption, enlarged liver and kidney, damage to thymus and sperm reservoir, diarrhea, skin inflammation, and fur loss in weanling rats fed heated corn and peanut oil; reduced antioxidant tocopherol in gastrointestinal tract of chicks fed thermally oxidized PUFA; reports of formation of cocarcinogens during heating of corn oil and promotion of chemically-induced mammary tumors.
Branched Chain Fatty Acids [3,4,16].	Ruminant fats, dairy products.	Individuals with genetic disorder Refsum's syndrome: neurological abnormalities resulting from inability to metabolize branched chain fatty acids.

[1] Berardi and Goldblatt, 1980
[2] Hui, 1996a
[3] Hayes, 1982
[4] Mattson, 1973
[5] Roine et al., 1960
[6] Phelps et al., 1965
[7] Lee et al., 1968
[8] Miller et al., 1969
[9] Hendricks et al., 1980a
[10] Hendricks et al., 1980b
[11] Yannai, 1980
[12] Boyd, 1973
[13] Frankel, 1984
[14] Artman, 1969
[15] Andrews et al, 1960
[16] Steinberg et al., 1971

6. Epidemiological Studies

Although the focus of this document is the environmental effects of spilled vegetable oils and animal fats, a brief discussion of the effects of these oils on human health is included for several reasons. First, the ENVIRON report submitted by the Petitioners incorrectly states that there are no accumulating or otherwise harmful components in animal fats and vegetable oils that are irritating, toxic, or carcinogenic; and that animal fats and vegetable oils are consumed safely by wildlife and humans. The large number of human health studies, many with a substantial population size, provide a significant data base for examining the effects of long-term oral exposure to fats and certain classes of fats or their components or degradation products.

Second, humans may be exposed to spilled non-petroleum and petroleum oils through several routes. Inhalation of harmful vapors and dusts or mists and aerosols is often a significant route of human exposure to spilled petroleum oils, though it is rarely an important exposure route of less volatile vegetable oils and animal fats.

Third, humans and many animals often handle chemicals by similar mechanisms in the body and exhibit similar toxic effects, a tenet underlying the frequent use of animal tests in evaluations of human health risk. For example, certain PAHs that are human carcinogens also cause cancer in laboratory animals and in fish and other aquatic organisms in the environment. Thus, the findings of epidemiology studies are relevant to the evaluation of mechanisms of toxicity in animals, particularly when the epidemiology studies are large enough to overcome statistical limitations that are found with smaller data sets.

a. Human Health. Although fat is a major component of the human diet, the consumption of high amounts of fat or certain types of dietary fats and oils has been associated with several chronic diseases (Hui, 1996a; FAO/WHO, 1994; Nelson, 1990; Katan *et al.*, 1995). In a number of epidemiology studies, the intake of dietary fat and some fat types (e.g., saturated fats, unsaturated fats, polyunsaturated fatty acids, trans-fatty acids, cholesterol) has been correlated with the incidence of coronary artery disease. Dietary fat consumption has been associated with the incidence of certain types of cancer, including mammary and colon cancer, presumably because dietary fat is acting as a cancer promoter. Dietary fat intake has also been linked to hypertension, diabetes, and obesity (Hui, 1996a). Other studies report that high dietary fat intake is related to altered immunity and altered steroid excretion and may affect bone modeling and remodeling.

In many animal and human studies, dietary fat intake has been linked to cardiovascular disease and atherosclerosis through its effects on the levels of cholesterol and triglycerides in plasma and the lipid composition of lipoproteins (Hui, 1996a). A 2% rise in risk of coronary heart disease has been predicted for every 1% increase in serum cholesterol. The American Heart Association, American Cancer Society, and National Cancer Institute have recommended lowering fat intake to 30% of total consumed calories in adults; the American Heart Association also recommends limiting the intake of polyunsaturated fatty acids to less than 10% of calories and replacing saturated acids with monounsaturated acids (USDHHS, 1990; FAO/WHO, 1994; Hui 1996a).

b. Comparison of Effects From Oil Spills With Human Consumption of Vegetable Oils and Animal Fats. The ENVIRON report, which was submitted by the Petitioners, draws incorrect comparisons between the human consumption of vegetable oils and animal fats and the environmental effects of oil spills. The effects on humans who consume small quantities of vegetable oils and animal fats in their foods cannot be easily translated to environmental effects produced by oil spills. These situations differ in many respects. A few of the differences are highlighted below:

- Differences in factors relating to the host organism: Sensitivity; humans may not be the most sensitive species. Species differences; while similarities in metabolism and biokinetic parameters exist between some species, it is often unclear how effects on humans can be translated to effects on fish. Differences in susceptibility; there are no controls for differences in genetics, age, life-stage, strain, gender, health, nutritional status, presence of other chemicals, or other factors inherent to the exposed organisms.
- Differences in dose-response relationships. It is unclear how dose-response relationship can be extrapolated from humans to other species, even if such information had been provided.
- Exposure. Exposure differs in route, frequency, and duration. Animals are exposed to large quantities of oil during an oil spill, and the exposure may be short-term or long-term. The animals may ingest the oil, or they may be exposed through their gills or skin. Humans consuming foods, however, are exposed to small quantities of oils for intermittent periods of time, and their exposure is via ingestion only.
- Differences in chemical composition. The composition of oils used in small quantities in processed foods may differ from the composition of the oils spilled in the environment, particularly when the oils are acted upon by chemical and biological processes in the environment.
- Environmental factors. The effects of oil in the environment depend on a wide variety of factors, including pH and temperature. These factors are different from those that affect humans consuming food oils.
- Effects. Effects, such as reduced egg hatchability or effects on molting, cannot be measured in humans.
- Ecosystems. Ecosystems, food webs, and predator-prey relationships can be affected by oil spills; these are not factors in determining human health effects.
- Statistical power of studies. Those epidemiologic studies with large numbers of people have demonstrated possible adverse effects from consumption of high levels of dietary fat or types of fat. Negative studies may indicate that too few subjects were included in the study or that confounding factors obscured the effect because of statistical limitations of the methodology.

7. Other Adverse Effects of Oil Spills

a. Aesthetic Effects: Fouling and Rancidity. Fouling of beaches and shoreline and rancid odors have been reported after spills of vegetable oils and animal fats; some real-world examples are provided in section II.D.2. Rancidity is the deterioration of fats and oils in the presence of oxygen (oxidative rancidity) or water (hydrolytic rancidity) with formation of off-flavors and odors (Hui, 1996b, 1996d; Kiritsakis, 1990). The hydrolysis and oxidation of spilled vegetable oils and animal fats and decomposition of hydroperoxides leads to formation of aldehydes, ketones, fatty acids, hydroperoxides, and other compounds that produce off-flavors and rancid odors. Rancidity occurs especially with oils that contain PUFA, such as linoleic acid (Hui, 1996a). Fish oils, which contain high levels of PUFA, are especially susceptible to oxidative rancidity and production of toxic byproducts and are often supplemented with antioxidants to reduce their oxidation.

Unlike vegetable oils and animal fats, rancid odors have not been reported following petroleum oil spills, although off-flavors and tainting of fish have occurred (Crump-Wiesner, 1975;

Hartung, 1995). Fish collected near petroleum refineries or in petroleum-polluted areas can be tainted (Lee, 1977), and commercial species have been contaminated with petroleum oils (Michael, 1977). Thousands of observations of floating tar balls and beach tar have been tabulated over a 4-year period in a petroleum monitoring project for marine pollution (NAS, 1985d).

b. Fire Hazards. While some petroleum oils and products present fire and explosion hazards, most vegetable oils and animal fats do not, unless flammable chemicals, such as hexane used during processing, are present or temperatures are elevated. A few vegetable oils, such as coconut oil (copra oil) are spontaneously combustible (Lewis, 1996). Because of their low vapor pressures, some petroleum products are highly volatile and flammable. In addition, most vegetable oils and animal fats have a high flash point (temperature at which decomposition products can be ignited), while the flash point for many petroleum products is below or near room temperature.

Although most vegetable oils and animal fats do not easily catch fire by themselves, once fires begin they are difficult to extinguish and may cause considerable environmental damage. For example, a butter and lard fire in Wisconsin that was apparently started by an electric forklift resulted in the release of some 15 million pounds of melted butter that threatened nearby aquatic resources (Wisconsin, 1991a, 1991b, 1991c; Wisconsin State Journal, 1991a, 1991b, 1991c, 1991d, 1991e).

c. Effects on Water Treatment. Oils and greases of animal and vegetable origin and those associated with petroleum sources have long been a concern in wastewater control (USEPA, 1979; Metcalf and Eddy, 1972). Too much oil, i.e., spills or discharges of oil and grease to a municipal wastewater treatment system in quantities that exceed the levels the treatment plant was designed to handle, can overwhelm the water treatment plant that maintains sanitary conditions and removes water pollutants that are harmful to aquatic organisms or interfere with the recreational value of waters (Institute, 1985; Metcalf and Eddy, 1972). Certain fatty acid products, such as quaternary amines, may inhibit biological treatment and affect in-plant facilities and downstream municipal sanitary sewage treatment facilities (Hui, 1996d).

Under normal operations, floating oil can be removed before wastewater is discharged to water treatment plants, and highly variable discharges of flow and organics can be minimized (Institute, 1985). With large quantities of spilled oil and high organic loads, however, these conditions may not be controlled adequately and water treatment systems can be damaged. To prevent potential damage to water treatment plants from oil spills, officials may halt water treatment and interrupt water supplies, as occurred when 15 municipal drinking water intakes were shut down following a spill of one million gallons of diesel fuel from a collapsed storage tank at the Ashland Oil facility in Floreffe, Pennsylvania in 1988 (USEPA, 1988).

8. FWS Comments

The FWS submitted a memorandum with the following position to the EPA in 1994. The potential for harm from petroleum and non-petroleum oils is equivalent; the path to injury is different. Edible non-petroleum oils cause chronic effects with the potential of mortality. Both petroleum and non-petroleum oil impact natural resources through the fouling of coats and plumage of wildlife. Secondary effects from fouling include drowning, mortality by predation, starvation, and suffocation. The removal of edible oil is more difficult and strenuous for wildlife due to the low viscosity of vegetable oil, which allows deeper penetration into body plumage or fur and thorough contamination of the wildlife.

Edible oils ingested in large quantities can cause lipid pneumonia. Edible oil consumed by wildlife during preening or cleaning of their coats also acts as a laxative resulting in diarrhea and dehydration. Small amounts of edible oil on plumage can cause thermal circulation troubles and embryo death in eggs exposed to oil through disruption of egg/air interface (USDOI/FWS, 1994).

C. Petitioners' Claim: Animal Fats and Vegetable Oils Are Essential Components of Human and Wildlife Diets

Petitioners claim that animal fats and vegetable oils are essential components of human and wildlife diets.

EPA Response: While EPA agrees that some components of animal fats and vegetable oils are essential components of human and wildlife diets, EPA disagrees with the Petitioners that all animal fats and vegetable oils are essential components of human and wildlife diets. Most species require only one or two essential fatty acids. Most animals need some level of fat to supply energy and fat-soluble vitamins. Intake of high levels of dietary fat, some types of fat, and essential fatty acids, however, can cause adverse effects.

While low levels of certain chemicals are essential for health, exposure to high levels of these chemicals produces toxicity. Numerous examples in the scientific literature demonstrate that essentiality does not confer safety and essential elements can produce toxic effects. Among these chemicals are vitamin A; the fatty acid a-linolenic acid, an essential fatty acid in humans and coldwater fish; and trace metals such as iron, manganese, selenium, and copper (Klaassen et al., 1986; NAS, 1977a; USEPA, 1980; Rand and Petrocelli, 1985; Abernathy, 1992; Hui, 1996a; NAS/NRC 1981a).

Further, high levels of fats and oils alter the requirements for essential fatty acids and change the balance between certain types of lipids and fatty acids. For many species of fish and laboratory animals, levels of essential fatty acids must be increased for the animals to tolerate high lipid levels (NAS/NRC, 1983, 1995). High levels of some fatty acids (n-6 PUFA, including the essential fatty acid linoleic acid) deplete other fatty acids (n-3 PUFA, including the essential fatty acid a-linolenic acid), thereby creating nutritional deficiency. In addition, constituents of vegetable oils and animal fats also affect requirements for essential fatty acids. Erucic acid, a constituent of rapeseed oil, adversely affects reproduction in rats by interfering with the metabolism of essential fatty acids (Roine *et al.*, 1960).

Animals often die from starvation after oil spills destroy their food supply by oiling food or making it unavailable. In addition to a reduction in food supply and a need to consume twice their normal amount of food to maintain body temperature (Hartung, 1965; 1995), oiled birds that are unable to float or fly cannot retrieve food from the water that usually provides their food. Bird rescuers have described dead birds with organs were filled with oil after eating oiled food or consuming oil while preening their feathers to remove oil (Croxall, 1975; Lyall, 1991; Frink and Miller, 1995). Thus, EPA finds that Petitioners' arguments are non-persuasive and have little relevance to the large quantities of oil released into the environment from oil spills.

1. Nutritional Requirements for Dietary Fat

In addition to their roles in cellular structure, membrane integrity, and microsomal enzyme function, fats play an important nutritional role by supplying energy and essential nutrients (Rechigl, 1981; Hui, 1996b; Van Soest,

1982). The caloric value of fats is more than twice that of carbohydrates or proteins (Hui, 1996a). Fats are a source of the fat-soluble vitamins A, D, E, and K and are rich in antioxidants, including tocopherols, such as vitamin E, and carotenes such as provitamin A. They also facilitate the digestion and absorption of vitamins.

The nutritional requirements for dietary fat vary greatly among species. A diet containing about 5% dietary fat is recommended for most laboratory animals (NAS/NRC, 1995). Growth usually increases greatly in animals fed a diet containing higher levels of fat, but lifespans are shortened and lactation performance and reproduction adversely affected in rats fed diets with 30% lipid (French *et al.,* 1953). In minks, diets with 35–40% fat have been satisfactory for meeting energy requirements, but higher levels (44–53%) are recommended for fur development, pregnancy and lactation (NAS/NRC, 1992.) Up to 44% fresh fat was used in fox diets without detrimental effects (NAS/NRC, 1992). For coldwater fish, 10% to 20% lipid is needed in diets, and higher levels of lipid alter carcass composition by deposition of excess lipid and reduction of the percentage of body protein (NAS/NRC, 1981a).

Nutritional requirements for fats are affected by environmental influences and the health status of the organisms. Birds must consume twice as much food after a spill for thermal regulation (Hartung, 1967). In laboratory animals, the requirement for certain fatty acids (n-6 PUFA) is increased during lactation (NAS/NRC, 1995).

For many animals (cattle, goats, and sheep), vitamin and energy requirements rather than specific dietary requirements for fat are enumerated (NAS/NRC 1981b; NAS/NRC, 1985; NAS/NRC, 1984). Certain types of fat are necessary for other animals. For example, sterols and perhaps lecithin are necessary for crustaceans (NAS/NRC, 1983).

Dietary Requirements of Wild Animals. Unlike domestic animals that are fed under regimens to maximize their productivity, wild animals and free-ranging domestic animals may have different nutritional requirements for their survival, growth, and reproduction (Van Soest, 1982). Diets that promote growth and obesity may also shorten life and are undesirable for wild animals.

2. Essential Fatty Acids (EFA)

Certain unsaturated fatty acids that must be supplied in the diet are called essential, because humans or other animals lack the enzymes to synthesize them (Hui, 1996a; Rechigl, 1983). Two fatty acids are considered essential in humans—linoleic acid and a-linolenic acid (Hui 1996a). These essential fatty acids are required for fetal development and growth. Long-chain n-3 polyunsaturated fatty acids, such as a-linolenic acid, are needed by the brain and retina; learning disabilities and loss of visual acuity have been observed in animals with low levels of these fatty acids. A balance of PUFA from both the n-6 and n-3 families is needed to maintain health (Hui, 1996a).

EFA requirements differ according to species. In chickens, 1% of the EFA linoleic acid is required; the essentiality of a-linolenic acid has not yet been proven for poultry (NAS/NRC, 1994). Linoleic acid is an EFA for pigs; arachidonic, which is generally added to swine diets, can be synthesized from linoleic acid (NAS/NRC, 1988). Minks require linoleic acid, and rabbits can develop EFA deficiency (NAS/NRC, 1992, 1977b). Silver foxes need 2 to 3 grams of EFA linoleic and linolenic acids daily to prevent skin problems and dandruff (NAS/NRC, 1992). The dietary EFA requirements of ruminants are about an order of magnitude lower than those of non-ruminants (Van Soest, 1982).

Studies of fish and crustaceans demonstrate that EFA requirements of aquatic animals vary with species and are apparently related to the ability of the animals to convert linolenic acid (18:3w3) to highly unsaturated fatty acids (Kanazawa et al., 1979). While some animals can synthesize necessary fatty acids, others require them in their diets. The n-3 fatty acids are essential for good health and growth in rainbow trout, red sea bream, and turbot (NAS/NRC, 1981a). For chum salmon, the requirement for linoleic and linolenic acids is 1%, or 0.5–1% for n-3 PUFA in the diet. For coho salmon, the optimal level of n-3 fatty acids is 1–2.5%, and the optimal level of n-3 plus n-6 fatty acids appears to be approximately 2.5%. EFA requirements can be affected by many factors, including fat content of the diet and temperature. In fish, EFA requirements change with temperature and culture conditions (NAS/NRC, 1983, 1981a.)

3. Adverse Effects of High Levels of EFAs

While certain levels of fat and essential fatty acids are necessary, higher levels can produce adverse effects. Although requirements for linolenic acid, a n-3 polyunsaturated fatty acid, are as high as 0.5% of total caloric intake in humans, consumption of a diet high in the same family of fatty acids (n-3 PUFA) may cause oxidative stress to cell membranes through lipid oxidation reactions, thereby increasing requirements for antioxidants (Hui, 1996a).

A balance of types of lipid and various fatty acids is needed. For example, many species of fish and laboratory animals tolerate high levels of lipid if the essential fatty acid levels are increased. (NAS/NRC, 1983, 1995). Similarly, a high level of other dietary components can increase the need for certain PUFAs (n–6 PUFA) in rats, and alter the fatty acid balance (between n–6 PUFA and n–3 PUFA) (NAS/NRC, 1995). High levels of some fatty acids (n–6 PUFA) deplete other fatty acids (n–3 PUFA), thereby creating adverse effects associated with nutritional deficiency.

Compared to rodents consuming diets high in saturated fatty acids, rodents receiving diets rich in linoleic acid—one of the two essential fatty acids for humans—exhibited increased development of breast tumors, including a shortened latency period for tumor appearance, promotion of tumor growth, and increased incidence of mammary tumors (Hui, 1996a). Once the dietary linoleic acid exceeded 4–5% of total calories, saturated or unsaturated fats linearly increased tumor incidence. Dietary linoleic acid enhanced the spread of mammary tumors to lungs in rats, apparently by acting as a cancer promoter. Fish oil, which contains n–3 PUFAs, inhibited mammary tumor development, apparently inhibiting the effects of linoleic acid.

The importance of balance in essential fatty acids is clearly seen in studies of coldwater fish. An optimum level of unsaturated fatty acids is required for maximum growth of coldwater fish, and the requirement for n–3 fatty acids may be species-specific (NAS/NRC, 1981a). EFA deficiency is characterized by poor growth as well as numerous other symptoms, and the deficiency of most symptoms can be reversed with certain fatty acids (n–3 PUFA); the addition of other fatty acids (n–6 PUFA) to the diet reverses some symptoms, while others are aggravated.

In coho salmon, extremely low and high levels of n–3 fatty acids inhibit growth; concentrations of n–6 fatty acids above 1% also depressed growth (NAS/NRC, 1981a). In studies of rainbow trout fed different levels of triglycerides containing n–3 and n–6 fatty acids in diets containing 10% lipid, growth was reduced when diets were deficient in n–3 fatty acids, high in n–6 and low in n–3 fatty acids, or high in both n–3 and n–6 fatty acids.

4. Adverse Effects of High Levels of Fats and Oils

Although fat intake is necessary to provide energy, vitamins, and EFA, ingestion of high levels of dietary fat can cause adverse effects in fish and aquatic species, other animals, and humans. The adverse effects of consumption of high levels of dietary fat and certain classes of fat by humans and animals have been discussed extensively in section II.C.3.

5. Relevance of EFA Principles to Spills

For most animals, only one or two fatty acids are essential, and these are not necessarily the fatty acids present in an oil spill. Animals require only small quantities of these EFAs that are provided in a normal diet, and these quantities must be in balance. While low levels of one or two fatty acids are needed by some species, in several species tested, high levels of these fatty acids produce adverse effects by toxicity or by creating nutrient imbalances that deplete other essential nutrients.

After a spill, high levels of animal fats and vegetable oils other than the EFA are present in the environment. High levels of total dietary fat, certain classes of fats, imbalances of types of fat, and some components and breakdown products produce adverse effects in laboratory animals and in some animals that have been examined in the field and are associated with adverse effects in humans. Further, some constituents of vegetable oils, such as erucic acid in cottonseed oil, actually interfere with EFA metabolism, thereby causing adverse effects (Roine et al., 1960).

When food is coated with oil from a spill of vegetable oils or animal fats, animals are unable to forage or consume the food or suffer the consequences of ingesting large quantities of oil as they consume food. Oil-coated birds die of hypothermia or starvation when they are unable to obtain or consume twice their normal amount of food to provide the increased metabolic requirements needed to survive oil spills.

Some oils, their constituents, or transformation products remain in the environment for years. By contaminating the food source biomass, reducing breeding animals and plants that provide future food sources, contaminating nesting habitats, and reducing reproductive success through contamination and reduced hatchability of eggs, oil spills can cause long-term effects for years even if the oil remains in the environment for relatively short periods of time.

6. FWS Comments on Essential Fatty Acids

The FWS commented that although fats and oils are used by cells of living organisms in small amounts, too much will cause harm to organisms through means other than toxicity. Ingestion of concentrated vegetable oil or animal fat could cause indigestion, nausea, and diarrhea. This could incapacitate a bird or mammal (USDOI/FWS, 1994).

D. Petitioners' Claim: Animal Fats and Vegetable Oils Are Readily Biodegradable and Do Not Persist in the Environment

EPA disagrees with Petitioners' claim that all animal fats and vegetable oils are readily biodegradable and notes that when biodegradation does occur in the environment, it can lead to oxygen depletion and death of fish and other aquatic organisms. Some products formed by biodegradation and other transformation processes are more toxic than the original oils and fats. While some animal fats and vegetable oils are degraded rapidly under certain conditions, others persist in the environment years after the oil was spilled (Mudge *et al.*, 1995; Mudge, 1995, 1997a, 1997b). Further, spilled animal fats and vegetable oils can cause long-term deleterious environmental effects even if they remain in the environment for relatively short periods of time, because they destroy existing and future food sources, reduce breeding animals and plants, and contaminate eggs and nesting habitats.

Every spill is different. How long the vegetable oil or animal fat remains in the environment after it is spilled, what proportion of the oil is degraded and at what rate, what products are formed, and where the oil and its products are transported and distributed are determined by the properties of the oil itself and those of the environment where the oils is spilled. Factors such as pH (acidity), temperature, oxygen concentration, dispersal of oil, the presence of other chemicals, soil characteristics, nutrient quantities, and populations of various microorganisms at the location of the spill profoundly influence the degradation of oil.

Like petroleum oils, vegetable oils and animal fats can float on water, settle on sediments or shorelines, and form emulsions when there is agitation or prolonged exposure to heat or light (Crump-Wiesner and Jennings, 1975; DOC/NOAA, 1992, 1996). Environmental processes can alter the chemical composition and environmental behavior of the spilled oils and influence their proximity to environmentally sensitive areas and the environmental damage they cause.

The detrimental environmental effects of several spills of vegetable oils and animal fats are described below and in Appendix I, Table 4: Effects of Real-World Oil Spills. These reports provide examples of the effects of some specific spills where death, injuries, and damage were observed. No structured survey on the effects and numbers of victims of spills of vegetable oils and animal fats has been conducted (Rozemeijer *et al.*, 1992). Because birds and other animals show only a ''wet look'' when they are coated with vegetable oils and animal fats, they are difficult to identify and may never be found if they sink when they die or are consumed by predators (NAS, 1985e).

1. Chemical and Biological Processes Affecting Vegetable Oils and Animal Fats in the Environment

Vegetable oils and animal fats that are spilled in the environment can be transported and transformed by a wide variety of physical, chemical, and biological processes that alter the composition of the oil, its fate in the environment, and its toxicity. Oil that is spilled in inland waters, such as small rivers and streams, may be especially harmful if there are limited oxygen resources in the water body and little dispersal of the oil (NOAA/FWS, 1996).

Whether the toxicity of these transformation products formed by chemical and biological processes increases compared to that of the original oil depends on the specific oil and the products that are formed. For example, lipid oxidation products that are formed following exposure of fats to oxygen, light, and inorganic and organic catalysts have been associated with colon cancer; and cholesterol oxidation products that are formed by autoxidation of cholesterol exposed to air, heat, photooxidation, and oxidation agents have numerous biological activities (Hui, 1996a). (See section II.B.5.d for a discussion of the toxicity of transformation products.)

a. Chemical Processes. The fate of petroleum and non-petroleum oils can be altered by environmental processes. Primary weathering processes include spreading, evaporation, dissolution, dispersion, emulsification, and sedimentation (DOC/NOAA, 1992a, 1994, 1996). The rate and relative importance of each of these processes depends on the specific oil that is spilled and environmental conditions that are present and that may change over time. Wind transport, photochemical degradation, and microbial degradation may also play

important roles in the transformation of petroleum oils, vegetable oils and animal fats.

Different parts of the ecosystem are affected as the composition of the spilled oil changes. For example, weathered petroleum oils penetrate into marsh vegetation less than fresh oil, for weathered oil is composed of relatively insoluble compounds and often forms mats or tarballs (DOC/NOAA, 1994; Hartung, 1995; NAS, 1985e). Thus, weathering decreases the potential exposure to fish through the water column while increasing the potential exposure of species that ingest tarballs. As the lighter fractions dissolve or evaporate, oil sinks, thereby contaminating sediments and contributing to water column toxicity. Spilled sunflower oil is hydrolyzed and polymerized to chewing gum balls that can be washed ashore or can sink and cover sediments, thereby exposing benthic and intertidal marine communities (Mudge, 1993).

Vegetable oils and animal fats can undergo several types of chemical reactions. They can be hydrolyzed to yield free fatty acids and diglycerides, monoglycerides, or glycerol; this hydrolysis can be catalyzed by acids, bases, enzymes, and other substances (Hui, 1996a; Lawson, 1995; Kiritsakis, 1990; Hoffmann, 1989). Vegetable oils and animal fats can be oxidized to form hydroperoxides and free radicals which perpetuate the oxidation reaction until they are destroyed by reacting with other chemicals, such as natural or added antioxidants. The free radicals that initiate an autoxidation reaction are formed by decomposition of hydroperoxides, exposure to heat or light, or other means. COPs are formed by autoxidation of cholesterol that is exposed to air, heat, photooxidation, and oxidative agents derived from dietary sources and metabolism (Hui, 1996a).

Several types of reactions can occur during processing, cooking, or storage of fats and oils, including hydrogenation of unsaturated fatty acids in oils (hardening); esterification; interesterification, including transesterification; and halogenation (Lawson, 1995; Hui, 1996a; Hoffmann, 1989; Yannai, 1980). Thermal oxidation and polymerization during cooking, frying, or processing operations at high temperatures, generally between 180°C to 250°C, can lead to conjugation (act of being joined) of polyunsaturated fatty acids and cylization and the formation of volatile decomposition products.

b. Biological Processes. Petroleum oils and vegetable oils and animal fats that are spilled in the environment can be transformed by bacteria, yeast, fungi, and other microorganisms. Although microbial degradation rarely occurs when there are controlled conditions during normal storage of animal fats and vegetable oils, microorganisms can grow on vegetable oils and animal fats and degrade them when environmental conditions are favorable (Ratledge, 1994).

Investigations of biological approaches to remediating sites contaminated with petroleum oils have shown that numerous environmental factors must be carefully controlled for biodegradation to be effective in reducing contamination from oily materials in soil (Venosa et al., 1996; Salanitro *et al.*, 1997). While bioremediation has been used for soil cleanup at some petroleum-contaminated sites (e.g., in tests at refineries, in treatment of oily sludges in oil and gas operations, and at pipeline sites for spills of crude oil), successful cleanup requires management of appropriate levels of applied waste to soil, aeration and mixing, nutrient fertilizer addition according to the ratios of carbon: nitrogen: phosphorus present, pH amendment, and moisture control to optimize degradation by soil micoorganisms (Salanitro *et al.*, 1997). The extent of biodegradation apparently depends upon the type of soil and crude oil involved.

The promise and the limitations of microbial degradation have been highlighted in numerous studies of factors influencing the microbial utilization of animal fats and vegetable oils (Ratledge, 1994). These studies were conducted in experimental cultures and cannot be applied readily to cleanups of oil spills, where control of pH, oil dispersal, and nutrient supplementation are difficult to achieve. They are described briefly, primarily to illustrate the complexity of biotransformation processes, the many factors that can affect biodegradation, and the difficulty in accurately reflecting conditions and determining rates of biodegradation or other transformation processes at specific spill locations. A more detailed discussion of the microbial degradation of vegetable oils and animal fats is provided in the accompanying Technical Document. (See Technical Document, Claims V and VI, Biological Processes, Section A.)

Factors that affect the biodegradation of oils include pH, dispersal of oil, dissolved oxygen, presence of nutrients in the proper proportions, soil type, type of oil, and the concentration of undissociated fatty acids in water. In addition to microorganisms, other biota can also alter the chemical composition of vegetable oils and animal fats. The reactions may depend on the species, for organisms such as invertebrates, lack enzymes that participate in certain metabolic pathways found in other organisms.

c. Rancidity. Biological and chemical processes can lead to the formation of rancid products that cause off-flavors and unpleasant odors. Rancidity results from the oxidation of unsaturated fatty acids that are acted upon by peroxide radicals or enzymes to form a variety of products, some of which are toxic (Hui, 1996a; Yannai, 1980). Rancidity can also be produced by hydrolysis of triglycerides and lipolysis by microorganisms or natural enzymes (Kiritsakis, 1990). The hydrolysis and oxidation of spilled vegetable oils and animal fats leads to formation of aldehydes, ketones, fatty acids, and other compounds responsible for off-flavors and rancid odors. The rate of rancidity increases with thermal decomposition of fats (Hui, 1996a), although enzymatic peroxidation and oxidation of unsaturated fatty acids by lipoxygenases can also occur in plant food stuffs even during storage at low temperature and in the dark (Yannai, 1980).

2. Environmental Fate and Effects of Spilled Vegetable Oils and Animal Fats: Real-World Examples

The reports in this section describe the spread of vegetable oils and animal fats after spills into the environment and detail the deleterious effects produced by these spills. While some aspects of specific spills have been discussed earlier, the examples presented below demonstrate that factors such as the nature of the oil, its environmental fate, and proximity of the spill to environmentally sensitive areas determine the adverse effects of spills of vegetable oils and animal fats in the environment. Many spills are never reported. Animals injured or killed by oils may never be found, for they are highly vulnerable to predators or may drown and sink (USDOI, 1994; Frink, 1994; NAS, 1985e). Thus, the reports that are summarized in Appendix I, Table 4 and below are not a comprehensive study of the adverse environmental effects of spills of vegetable oils and animal fats, but rather a snapshot revealing some of the deleterious effects caused by spills of oil into the environment.

Minnesota Soybean Oil and Petroleum Oil Spills. Oil from two spills in Minnesota killed thousands of ducks and other waterfowl and wildlife or injured them through coating with oil. The peak of waterfowl damage occurred

within two days of the breakup of ice on the Minnesota and Mississippi rivers in the spring of 1963 (Minnesota, 1963; USDHHS, 1963). There were two sources of oil—an estimated 1 million to 1.5 million gallons of soybean oil that entered the Minnesota River via the Blue Earth River when storage facilities failed at a plant in Mankato, Minnesota; and an estimated 1 million gallons of low viscosity cutting oil that escaped to the Minnesota River near Savage, Minnesota, from a marsh that was flooded with oil when storage facilities failed. Oil spilled during the winter months from mechanical failure of storage tanks or pipelines, moved little until the breakup of ice in the spring. The varnish-like covering of willows on the river banks showed that the soybean oil had escaped into the river during the spring run-off.

While the petroleum oil and soybean oil slicks could not be distinguished by field observation, laboratory analysis of samples of oil and oil scraped from ducks revealed that soybean oil caused much of the waterfowl loss (Minnesota, 1963). Approximately 5,300 birds were affected or killed by oil, including 1369 live oil-soaked ducks rescued and 1842 dead birds collected. They included lesser scaup ducks, ringnecked ducks, coots and grebes, several other types of ducks, gulls, and mergansers, and a cormorant. While some birds may have been counted more than once, the numbers probably underestimate the impact of the oil spills, because ducks covered with oil crawl into dense cover and are hard to find.

Mammals and other dead animals were reported, including about 26 beaver, 177 muskrats, and 50 others, among them turtles, herons, kingfisher, songbirds, other birds, skunk, squirrel, dog, and cows (Minnesota, 1963). The death of 7,000 fish was attributed to causes other than oil pollution, because winterkill is common in shallow backwater areas of the river and a BOD study indicated that the sample analyzed would not have sufficient oxygen demand to significantly affect oxygen resources in the river. Bottom fauna used as fish food may have been affected temporarily in localized areas.

The character of the soybean oil on and in the water changed with time, as thick orange-colored slicks that were first observed changed to pliable greyish and somewhat rubbery floating masses that were stringy or somewhat rounded and were sometimes surrounded by a light oil slick (Minnesota, 1963). Limited areas of the bottom were covered.

Oil that normally floated on the surface of the river tended to sink to the lake bottom or settled into low areas of the river bottom near the shoreline, apparently because of entrapment of heavy materials in the oily mass. A sample of soybean oil collected from the bottom of the lake contained sand, dirt, twigs, and leaves when it was analyzed in the laboratory.

Soybean oil also mixed with sand on the beach, creating a hard crust 3 feet above water level. White balls, apparently from soybean oil that was once near the surface of a lake, moved toward shore and broke up into long, white stringy material that collected on shore. Pools of tough, milky material covered with brown scum were found in low areas of the beach along with a hard varnish-like crust on the beach.

Spill of Coconut Oil, Palm Oil, and Edible Materials. In 1975, a cargo ship that was carrying primarily vegetable oils and edible raw materials (copra or dried coconut meat, palm oil, coconut oil, and cocoa beans) went aground on Fanning Atoll, Line Island and dumped its cargo onto a pristine coral reef (Russell and Carlson, 1978). The effects of the oily substances were similar to those following a petroleum oil spill. Fish, crustaceans, and mollusks were killed. Shifts in the algal community were observed, with excessive growth of some types of green algae and the elimination of other algal competitors. The effects on the algal community continued for about 11 months.

Sunflower Oil Spill in North Wales. When a cargo of unrefined sunflower oil was spilled into the environment off the coast of Anglesey, North Wales in January 1991, surface slicks of the oil were formed for many miles around the ship (Mudge *et al.,* 1993; Salgado, 1992, 1995). Some oil was hydrolyzed and polymerized to form ''chewing gum balls'' that were washed ashore over a wide area. The denser balls sank, allowing the sunflower oil to contact a wide range of benthic and intertidal communities near the spill. Sunflower oil polymerized in seawater and formed lumps that could not be degraded by bacteria.

Mussels that were near the spill died. Polymerized sunflower oil formed a cap that reduced the permeability of sediments to water and oxygen and killed organisms living on the sediments (Mudge et al., 1993, 1995, Mudge, 1995). Polymerization of sunflower oil that washed ashore produced concrete-like aggregates that still persist nearly 6 years after the spill (Mudge, 1997a, 1997b).

Rapeseed Oil Spills in Vancouver Harbor. Three small spills of rapeseed oil caused greater losses of birds than 176 spills of petroleum oils over a 5-year period in Vancouver harbor from 1974 to 1978 (McKelvey et al., 1980). An estimated 35 barrels of rapeseed oil killed an estimated 500 birds, while all of the petroleum oil spills combined oiled less than 50 birds, perhaps because the vegetable oils lacked the strong, irritating odor of petroleum or its eye-catching iridescence. Both petroleum and non-petroleum oils coat the feathers of birds, destroying their waterproofing qualities and allowing water to penetrate to the skin with loss of insulation and buoyancy, which results in exposure, and death (Mudge, 1995; Hartung, 1967; NAS, 1985e; Smith and Herunter, 1989; Rozemeijer, 1992).

Another spill of rapeseed oil (Canola) occurred in Vancouver Harbor on February 26, 1989 (Smith and Herunter, 1989). During product transfer, an estimated 400 gallons of rapeseed oil spilled into the harbor. A thin film covered large portions of the harbor, and a patchy slick of yellow oil from the spill site to the center of the harbor was visible from above. It was estimated that at least 700 birds were in the harbor at the time of the spill, including 500 diving ducks, 100 gulls, and 100 other divers.

Initially, booms were not used to contain the spill, and an attempt to disperse the oil with multiple passes of a small tug through the thick oil were ineffective (Smith and Herunter, 1989). EPA notes that the trade association requested that this ineffective mechanical dispersal be allowed as a response to spills of vegetable oil and animal fat under the FRP rule. After several hours, booms were set up to contain the oil and skimmer boats recovered the oil.

Cleanup was concluded 15 hours after the spill was discovered (Smith and Herunter, 1989). Nevertheless, 88 oiled birds of 14 species were recovered after the spill, and half of them were dead. Oiled birds usually are not recovered for 3 days after a spill, when they become weakened enough to be captured. Of the survivors, half died during treatment.

The authors caution that because vegetable oils are edible, they may not be considered as threatening to aquatic birds as petroleum oils. However, the end result is the same. Birds die (Smith and Herunter, 1989). The number of casualties from the rapeseed oil spills was probably higher than the number of birds recovered, because heavily oiled birds sink and dying or dead birds are captured quickly by raptors and scavengers.

Smith and Herunter emphasize that containing and recovering the spilled oil as soon as possible is critical to minimizing environmental damage

(1989). Using booms, testing transfer lines, having spill detection equipment in place, training on-site personnel, and reporting spills immediately are essential to reducing environmental harm.

Fat and Oil Pollution in New York State Waters. Pollution of surface waters by oils and fats from a wide variety of sources killed waterfowl, coated boats and beaches, tainted fish, and created taste and odor problems in water treatment plants in New York State (Crump-Wiesner and Jennings, 1975). Sources of the fats and oils included spills, food and soap manufacturing, refinery wastes, construction activities, industrial waste discharges, and sanitary sewage. Grease-like substances were seen along the shore or floating in Lake Ontario. Grease-balls that contaminated the shoreline near Rochester and smelled like fat or lard were analyzed and characterized as mixtures of animal and vegetable fats with similar fatty acid contents.

Spills of Fish Oil Mixtures in South Africa. Oil that was discharged from a fish factory effluent pipe near Bird Island, Lamberts Bay, South Africa, the breeding ground for 5,000 pairs of Cape Gannets and home to tens of thousands of Cape Cormorants and 500 Jackass Penguins, killed at least 709 Cape Gannets, 5,000 Cape Cormorants, and 108 Jackass Penguins (Percy Fitzpatrick Institute, 1974). A few days after the oiling incident, researchers found penguins covered with a sticky, white, foul-smelling coat of oil. They were shivering on the shore and gannet chicks, who were observed walking straight into the oil, were dead or dying. They observed a milky white sea on one side of the island and a frothy mixture and clots of oil thrown up on the island. The oil smelled strongly of fish.

Damage from fish-oil pollution was detailed at two other fish factories in South Africa (Newman and Pollock, 1973). In the rock lobster sanctuary at St. Helena Bay, 10,000 rock lobsters and thousands of sea urchins were killed, probably from oxygen depletion caused by the release of organic material from the fish factory. At least 100,000 clams died near a fish factory at Saldanha Bay along with large numbers of black mussels and prawns and some polychetes and anemones. Other effects were also described by the authors: the sea was discolored and smelled, water quality was poor, and the aesthetic appeal of the beaches located near a town and popular camping site was adversely affected.

Spill of Nonylphenol and Vegetable Oils in the Netherlands. Thousands of seabirds, mostly Guillemots and Razorbills, washed ashore in the Netherlands during a four-month period from December 1988 to March 1989 (Zoun, 1991). They were covered with an oil-like substance. Nearly all of the 1,500 sick birds that were taken to bird hospitals died; many exhibited emaciation, aggressive behavior, bloody stools, and leaky plumage. Autopsies and pathological examination of 30 birds revealed hepatic degeneration and necrosis as well as aspergilliosis in the air sacs and lungs. Chemical analysis of the feathers and organs showed the presence of high levels of nonylphenol and vegetable oils, such as palm oil. No source of the contaminants was established, but they may have been discharged from a ship.

Soybean Oil Spills in Georgia From a Tanker Truck and a Vegetable Oil Refinery. Aesthetic effects were a major concern to property owners on an oiled cove at Lake Lanier, Georgia (Rigger, 1997). The strong, unpleasant odor of soybean oil spilled from a tanker truck became more rancid as the oil weathered. Rapid response action minimized the damage and costs, although the oil adhered to boat dock floats and boats and produced several thousand dollars in claims for cleaning boats and docks and replacing dock floats.

In a vegetable oil refinery in Macon, Georgia, soybean oil was released from an aboveground storage tank that was accidentally overfilled (Rigger, 1997). Rapid response prevented significant damage from the spilled oil, which had flowed through a storm water system and entered a stream. Investigation of the spill incident revealed that previous spills from the facility had entered the sanitary sewer system and damaged the sewage treatment plant.

Wisconsin Butter Fire and Spill. In 1991, a major butter and grease fire apparently triggered by an electric forklift destroyed two large refrigerated warehouses at Central Storage facility in Madison, Wisconsin and resulted in the release of large volumes of butter, lard, cheese, meat, and other food products (Wisconsin, 1991a, 1991b, 1991c; Wisconsin State Journal, 1991a, 1991b, 1991c, 1991d, 1991e). The warehouses contained 15 million pounds of butter—much of it part of the USDA surplus program. Thick, black smoke filled the air, and melted butter and lard streamed from the burning building and threatened to pollute a nearby creek and lake.

The quick action of firefighters, city engineers, and other responders was credited by the company and state environmental officials with saving a nearby creek and lake from environmental disaster and limiting the losses and injuries from the fire (Wisconsin, 1991; Wisconsin State Journal, 1991a, 1991b, 1991c, 1991d, 1991e). If the buttery material had flowed through storm sewers into the creek and lake, it could have depleted the available oxygen required by walleyed pike, bass, and other aquatic organisms living in the creek and connecting lake and ruined a recent one million dollar cleanup effort in the watershed.

After the cleanup was largely completed, the Wisconsin Department of Natural Resources declared as hazardous substances the thousands of gallons of melted butter that ran offsite and the mountain of damaged and charred meat products spoiling in the hot sun and creating objectionable odors. The Wisconsin DNR stated that these products posed an imminent threat to human health and the environment.

3. FWS Comments on Degradation

Vegetable oils and animal fats may biodegrade quicker than petroleum; however, in the short term, this advantage is neutralized by the ability of many petroleum compounds to evaporate quickly. In addition, the higher BOD of vegetable oils and animal fats pose an increased risk of oxygen depletion in shallow waters and wetlands. Both kinds of oil will degrade more slowly in low-energy waters and can become submerged in an anoxic aquatic habitat, settle to the bottom and into sediments, or form thick layers because the vegetable oil is no longer being exposed to oxygenated waters or surroundings. In such instances, the edible oil or fat will remain in the environment for a long period of time and continue to create a risk to the natural environment. The variability of circumstances surrounding each spill (location, spill volume, weather, tides, water currents, effectiveness of spill response) will have a greater influence in the short term on environmental effects than will biodegradability. (USDOI/FWS, 1994)

E. Petitioners' Claim: Vegetable Oils and Animal Fats Have a High BOD, Which Could Result in Oxygen Deprivation Where There Is a Large Spill in a Confined Body of Water

Petitioners claim that vegetable oils and animal fats have a high BOD, which could result in oxygen deprivation where there is a large spill in a confined body of water with low flow and dilution.

EPA Response: EPA agrees with the Petitioners' claim that vegetable oils and

animal fats have a high BOD, which could lead to oxygen depletion and severe environmental consequences. (For a detailed discussion of this topic, see section II.B.4.a.Suffocation.) EPA disagrees, however, that oxygen depletion would occur only with large oil spills. Small spills are sufficient to cause oxygen depletion and suffocation and death of fish and other biota, depending on the conditions that apply at the location of the spill. Oxygen depletion can result from reduced oxygen exchange across the air-water surface below the spilled oil or from the high BOD by microorganisms degrading oil (Crump-Wiesner and Jennings, 1975; Mudge, 1995). Examples of environmental damage produced by small spills of vegetable oils and animal fats are provided above.

While a higher BOD is associated with greater biodegradability, it also reflects the increased likelihood of oxygen depletion and potential suffocation of aquatic organisms under certain environmental conditions (Crump-Wiesner and Jennings, 1975). Oxygen depletion and suffocation are produced by petroleum and vegetable oils and animal fats. Under certain conditions, however, some vegetable oils and animal fats present a far greater risk to aquatic organisms than other oils spilled in the environment, as indicated by their greater BOD.

According to studies designed to measure the degradation of fats in wastewater, some food oils exhibit nearly twice the BOD of fuel oil and several times the BOD of other petroleum-based oils (Groenewold, 1982; Institute, 1985; Crump-Wiesner and Jennings, 1975). While the higher BOD of food oils is associated with greater biodegradability by microorganisms using oxygen, it also reflects the increased likelihood of oxygen depletion and suffocation of aquatic organisms under certain environmental conditions (Groenewold, 1982; Institute, 1985; Crump-Wiesner, 1975). Oil creates the greatest demand on the dissolved oxygen concentration in smaller water bodies, depending on the extent of mixing (Crump-Wiesner and Jennings, 1975).

FWS Comments on BOD. Decomposition of vegetable oils and animal fats causes oxygen depletion problems for aquatic species (USDOI/FWS, 1994).

F. Petitioners' Claim: Vegetable Oils and Animal Fats Can Coat Aquatic Biota and Foul Wildlife

EPA Response: EPA agrees with the Petitioners' claim that vegetable oils and animal fats can coat aquatic biota and foul wildlife but disagrees with the lack of significance accorded this potentially devastating effect in Petitioners' ENVIRON report. Many animals and plants die when they are coated with spilled petroleum oils or vegetable oils and animal fats. (See section II.B.4.a. Coating with Oil for a discussion of these effects.) Coating with oil can contaminate existing and future food sources, destroy habitat, and damage eggs and nesting areas, thereby inflicting environmental damage years after an oil spill occurs (Frink and Miller, 1995).

Trustees Comments on Fouling. The biggest oversight of the ENVIRON report, which was never subject to peer review as are journal publications, is the insignificance given to the fouling potential of vegetable oils and animal fats (USDOI/FWS, 1994). Wildlife rehabilitators consider edible oils and fats to be some of the most difficult of substances to remove from wildlife because of their low viscosity. These less viscous oils are good wetting agents, allowing deeper penetration into plumage or fur and creating a thoroughly contaminated animal, as opposed to surface and intermediate penetration. In many instances, complete removal can only be accomplished with extremely hot water, which is detrimental because of scalding, and excessive washing.

The FWS takes issue with statements in the ENVIRON report that observed birds clean themselves and return to feeding areas (USDOI/FWS, 1994). Such observations are difficult to confirm without banding or radio tagging the birds and closely observing them. It is highly doubtful that the birds were able to clean themselves, for only minuscule amounts of oil can be completely preened from plumage. Even birds fouled with petroleum oils will preen and fly back to their nests. Small amounts of oil on the birds' plumage can cause thermal circulation trouble and smother embryos in eggs exposed to the oil. Birds may appear to act normally, but it is not the immediate effects of the oils but those that appear later that cause problems. Secondary effects from fouling include drowning, mortality by predation, starvation, and suffocation.

Both petroleum and non-petroleum oils foul the coats and plumage of wildlife (USDOI/FWS, 1994). The risks from vegetable oils and animal fats are magnified by their lack of repugnant smell or iridescence to frighten wildlife away, making it more likely that wildlife will come in contact with these oils.

III. Petitioners' Suggested Language To Amend the July 1, 1994, Facility Response Plan Rule

This section begins with a short discussion about EPA's inland area of jurisdiction and also provides some characterization of the amounts of vegetable oil and animal fats produced or consumed, and reported spills. These discussions are followed by EPA's response to the Petitioners' specific regulatory language to amend the July 1, 1994, facility response plan rule.

A. Background

Examples of water systems that occur in the inland area within EPA's zone of authority are major freshwater rivers, smaller streams, creeks, lakes and wetlands or mixed freshwater—saltwater estuary and wetlands areas subject to tides. (See a Memorandum of Understanding [MOU] between the Secretary of Transportation and the EPA Administrator dated November 24, 1971 [36 FR 24080].) Many of these areas, including wetlands and estuary areas, are often very sensitive, highly productive areas where a large number of organisms such as shrimp, crabs, fish, and water fowl nest, breed and feed. Lakes and larger rivers may be used as water supplies and have drinking water and industrial intakes that must be protected. Inland spills have a much higher potential to contaminate both ground and surface water supplies. Some lakes, estuaries and bays are often highly developed with industry, recreational beaches, marinas and other highly visible areas that need protection from oil spills.

Vegetable oil and animal fat were among the most frequently spilled organic materials, ranking sixth and seventh respectively, and were responsible for over 6% of all spills (384 of 6076 spills) of organic materials reported along the coasts and major waterways in the United States in 1973–1979 (Wolfe, 1986). Other authors estimate that at least 5% of all spill notifications are for vegetable oils and animal fats (Crump-Wiesner, 1975). Of the 18,000 to 24,000 spills in the United States reported annually to the National Response Center and EPA Regions, 2–12% are from non-petroleum oils, including vegetable oils and animal fats (USEPA/ERNS, 1995, 1996). These figures represent the minimum number of spills; it is likely that they greatly underestimate the actual number of spills because of significant underreporting. A comparison was made of reports of spills in Ohio of vegetable oil and soybean oil from January, 1984 to June, 1993 to the State

of Ohio Environmental Protection Agency (Ohio EPA) and to the National Response Center (NRC). Only 7 of 27 reports (26%) to the Ohio EPA were also reported to the NRC (USEPA, 1994a). There were a number of reports of vegetable and soybean oil spills to the NRC that were not on the State list (USEPA, 1994a).

B. Regulatory Language Changes Proposed by the Petitioners

Language to further clarify the definition of vegetable oil and animal fats. EPA Response: EPA has decided not to incorporate Petitioners' proposed definitions of ''animal fat and vegetable oils'' in the regulatory provisions of section 112.2. In issuing the final FRP rule, EPA included a definition of ''non-petroleum oil'' in an Appendix to the rule. (See 40 CFR part 112, Appendix E, section 1.2.3.) ''Non-petroleum oil'' is defined to mean ''oil of any kind that is not petroleum-based. It includes, but is not limited to, animal and vegetable oils.'' *Id*.

EPA included this definition of ''non-petroleum oil'' in the rule because the Agency established different and more flexible response planning requirements for facilities that handle, store, or transport non-petroleum oil, including animal fats and vegetable oils. For example, in calculating required response resources for non-petroleum facilities, the owner/operator of such a facility, including those facilities which handle, store, or transport animal fats or vegetable oils, is not required to use emulsification or evaporation factors in Appendix E of the rule. Rather, these facilities need only: (1) Show procedures and strategies for responding to the maximum extent practicable to a worst case discharge; (2) show sources of equipment and supplies necessary to locate, recover, and mitigate discharges; (3) demonstrate that the equipment identified will work in the conditions expected in the relevant geographic area, and respond within the required times; and (4) ensure the availability of required resources by contract or other approved means. 40 CFR Part 112, Appendix E, section 7.7. Importantly, EPA does not prescribe the type or amount of equipment that preparers of response plans for non-petroleum oil discharges must identify. *Id*.

Moreover, at the time of issuing the final rule, EPA also set forth definitions for both ''animal fat'' and ''vegetable oil'' in the preamble to the FRP rule (59 FR 34070, 34088 (July 1, 1994)). To assist owners and operators in distinguishing between oil types, EPA defined ''animal fat'' to mean ''a non-petroleum oil, fat, or grease derived from animal oils not specifically identified elsewhere.'' *Id*. The Agency defined ''vegetable oil'' to mean ''a non-petroleum oil or fat derived from plant seed, nuts, kernels or fruits not specifically identified elsewhere.'' *Id*. The Agency stands behind these definitions, and because EPA is not modifying the FRP rule as requested by Petitioners (see below), the Agency sees no need to include these definitions in the rule provisions.

Petitioners express a concern that animal fats and vegetable oils have been included with other types of ''non-petroleum oils,'' although the planning requirements for owners and operators of *all* facilities storing ''non-petroleum'' oils are more flexible than those requirements for facilities storing, handling, or transporting petroleum oil. Petitioners' main concern appears to be premised upon the claim that vegetable oils and animal fats are ''non-toxic'' compared to other non-petroleum oils. EPA believes that Petitioners have failed to make a demonstration that animal fats and vegetable oils should be subject to less stringent planning requirements than other types of non-petroleum oils. This is so for all of the reasons set forth elsewhere in this notice.

Allow mechanical dispersal and ''no action'' options to be considered in lieu of oil containment and recovery devices specified for response to a worst case discharge of vegetable oil and animal fats. EPA Response: The Agency declines this proposed language. Although the ''no action'' and mechanical dispersal options proposed by the Petitioners may be considered in response to an actual spill under certain conditions, i.e., river currents too high for the effective use of a boom, neither option would meet the intent of OPA for planning purposes. The intent of OPA was for industry to plan for and secure the equipment and resources needed to respond to a worst case discharge, which may be a discharge of 1 million gallons or greater for a large vegetable oil facility.

A ''no action'' plan would allow a large amount of oil to remain in the environment, which would in turn cause immediate physical effects to resources that could extend for considerable distances as the oil spreads. This oil would have the potential to remain in the environment for long periods of time.

One issue raised by the Petitioners is that the response to a spill of vegetable oil or animal fat may do more harm to the environment than a ''no action'' alternative. A consideration in the response to any type of oil, including petroleum or vegetable oil or animal fat, is whether the measures used in response to the spill will cause unacceptable damage to a specific type of environment. This determination is based on the conditions existing at the time of the spill. Specific spill conditions will often dictate the need for different techniques for the same water environment or shoreline habitat. A study, which evaluated the relative impact of various generic characteristics of response techniques in the absence of oil, rated booming and skimming as having a ''Low'' impact in open water, small lakes/ponds, large rivers and small rivers and streams (DOC/NOAA, 1992) and therefore, causing little environmental harm.

Mechanical dispersal of the vegetable oil or animal fat into the water column could shut down or negatively impact drinking intakes due to flavor changes and odors, reduce cooling efficiency in cooling waters of power plants, contaminate food from receiving waters, increase BOD levels, violate water quality standards, cause sludges, and adversely impact benthic organisms and the resulting food chain in inland areas. Oil dispersed by mechanical means may resurface and cause further environmental damage in the same area or a different area depending on the characteristics of the water body. (See section II.D.2, Rapeseed Oil Spills in Vancouver Harbor on the ineffective use of mechanical dispersal.) This Notice references studies that document spills of vegetable oils that have remained in the water environment for several years and that continued to kill shellfish and other organisms.

Limit the use of containment boom to the protection of fish and wildlife and sensitive environments: EPA's Response. Based on tests and studies summarized in the data in this Decision Document and the Technical Document, vegetable oils and animal fats clearly have adverse impacts on the aquatic and terrestrial environment and its inhabitants. EPA declines to modify the FRP rule as suggested by the Petitioners. EPA continues to believe that an OPA required FRP must limit the impacts of the oil through response techniques that include containment and removal in addition to protection of priority fish and wildlife and environmentally sensitive areas.

The Area Contingency Plan (ACP) identifies and prioritizes the fish and wildlife and environmentally sensitive areas to be protected and also determines the type of protection to be used when a spill occurs. CWA section 311(j)(5)(C)(I) requires that a FRP must be consistent with the applicable ACP, which usually requires that a

containment boom be positioned to protect drinking water intakes and environmentally sensitive areas.

In addition, facility response planning must also include the use of measures appropriate to the body of water to contain and limit and concentrate the spread of oil for removal. The spreading rate of oil is a function of its viscosity. Low viscosity materials spread easily over the surface of water. At lower temperature, the oil spreads less rapidly. Generally, vegetable oils and petroleum oils are of low viscosity. The spread of spilled oil over a large area will hamper recovery of the oil. The thicker the concentration of animal fat or vegetable and petroleum oil in an area, the greater the efficiency for oil removal. As the oil spreads over time into thinner slicks, its removal becomes less efficient and more costly. In tidally influenced areas, oil may move back and forth with each tide and be redeposited on the shore line, tidal flats, and marshes and cause adverse effects.

Since vegetable oils and animal fats usually have few volatile fractions and therefore usually do not decrease in volume through evaporation as do many of the lighter factions of petroleum oils, most of the quantity of vegetable oil and animal fats spilled into water remain in the environment. When this happens, there is the potential for adverse impacts to environmentally sensitive areas and water intakes. Although most vegetable oils and animal fats break down more quickly than some petroleum oils, under certain conditions and times of the year, these oils may remain in the aquatic environment for long periods of time, polarize and form toxic degradation products and kill shellfish and other organisms.

If a facility storing animal fat and/or vegetable oil does not provide for the use of containment booms in its plan to respond to a worst case discharge, it will not have the equipment and trained personnel available for an actual spill and many miles of shoreline and aquatic resources over a large area of water may be impacted. Rapid and immediate response and removal, including the use of containment booms, offer the most effective means of minimizing the immediate and long term effects of spills of petroleum and non-petroleum oils, including vegetable oils and animal fats. EPA does not believe that the Petitioners have shown why the use of containment booms should be limited to only protecting fish and wildlife and environmental sensitive areas. Without the use of containment booms, a worst case discharge of vegetable oil or animal fats could cause harm not only to fish and wildlife and environmentally sensitive areas, but also damage the aquatic and terrestrial environment. Such a discharge could also present risks to humans if the vegetable oil and animal fats adversely affect drinking water intakes.

Increase the time for the arrival of on-scene response resources for medium discharges and worst case Tier 1 response resources to 24 hours plus travel time from the currently required 12 hours including travel arrival time: EPA's Response. A rapid response to an oil spill is important in the recovery of as much oil product as possible. Any oil that remains in the environment will continue to adversely impact the aquatic and shoreline environment and cause lasting damage. (This document contains discussions of environmental, physical and other impacts that occur when vegetable oil and animal fats are spilled.) A 24 hour plus travel time delay in the arrival of response resources would result in an unacceptable increase in impacts to drinking water intakes, fish and wildlife and sensitive environments, greater response costs, less product recovered, and increased water and other types of pollution.

A delay in the arrival of response resources will increase the difficulty of the removal of the spilled oil and will also result in an increase in the cost to recover this oil. If effective containment and cleanup procedures are initiated within an hour of a spill occurrence, estimated removal costs are $250 per barrel (42 gallons). If two or more hours elapse before the oil is removed, the cost can be four or more times that amount and continue to increase with the time to respond to the release (USEPA, 1995). The ''window of opportunity'' for the most effective and efficient response to oil spills occurs within the early hours after the spill.

Immediate action is required when oil spills occur on water to prevent the oil from becoming so widely spread that containment and cleanup become extremely expensive and a larger area of fish and wildlife and environmentally sensitive areas are adversely affected. There are immediate physical effects to the environment from releases of vegetable oil and animal fat. There is the potential for additional sensitive areas to be contaminated within the 24 hours plus travel time proposed by the Petitioners for the arrival of response resources. This is 12 hours plus travel time longer than the FRP requirement for rivers, canals, inland, and near shore areas. Sensitive areas within many additional miles would be affected with the delay in the arrival of response resources proposed by the Petitioners since booms would not be made available for their protection until much later. Rapid response is imperative to limit adverse effects, protect resources, and contain oil for removal.

Extending the time for arrival of response resources would increase the FRP distance calculation for a facility and could result in additional vegetable oil and animal fat facilities meeting the criteria for substantial harm and having to prepare and submit a facility response plan to EPA. The requirements for determination of substantial harm in the FRP rule for facilities with 1 million gallons or above capacity includes a calculation in Appendix C–III of 40 CFR Part 112 of the distance an oil discharge from the facility would travel within the time it would take for the appropriate tier of response resources to arrive. Once the distance is calculated, the facility must determine whether fish and wildlife and environmentally sensitive areas or drinking water intakes are located within this distance. If so, the facility is considered a substantial harm facility and must prepare and submit a response plan. An additional twelve hours plus travel response time would more than double the distance a spill could travel on water before the arrival of response resources and therefore potentially increase impacts to drinking water intakes and environmentally sensitive areas and increase the number of vegetable oil and animal fat facilities that have to prepare and submit FRPs. For the above reasons, EPA declines to modify the FRP rule in this manner.

IV. Conclusions

The environmental effects of petroleum and non-petroleum oils, including vegetable oils and animal fats, are similar because of physical and chemical properties common to both. Many of the most devastating effects of spills of petroleum oils and vegetable oils and animal fats are physical effects, such as coating of animals, suffocation, or starvation. Some tests measuring BOD suggest that certain vegetable oils and animal fats may present a greater environmental risk of suffocation to organisms than spilled petroleum oils under certain conditions. Petroleum oils and vegetable oils and animal fats can be transferred to the eggs of nesting birds from the parents' feathers and smother the embryos inside. Embryos in eggs are also killed by petroleum oils through mechanisms of toxicity; whether non-petroleum oils also cause direct embryotoxicity has not been evaluated in tests.

Petroleum oils and vegetable oils and animal fats, can enter all parts of the

aquatic environment and adjacent shoreline. They can form a layer on water, settle on the bottom in sediments, foul shorelines, and be transported and distributed to other areas.

Some vegetable oils and animal fats, their components, or breakdown products remain in the environment for years. Whether or not the oil persists in the environment, spilled oil can have long-lasting deleterious environmental effects. By contaminating food sources, reducing breeding animals and plants that provide future food, contaminating nesting habitats, and reducing reproductive success through contamination and reduced hatchability of eggs, oil spills can cause long-term effects years later even if the oil remains in the environment for relatively short periods of time.

In addition to physical effects and the destruction of food and habitat, petroleum oils and vegetable oils and animal fats, their constituents, or degradation products can cause short-term and long-term toxic effects in some animals. Petroleum oils contain PAHs and benzene which are animal and human carcinogens. While vegetable oils and animal fats contain only small quantities of PAHs, high dietary intake of fats and certain types of fats have been associated with increased cancer incidence in laboratory animals and humans as well as coronary artery disease, diabetes, obesity, and altered immunity and other effects. Lethality, impaired growth, reproductive effects, and behavioral effects are among the subchronic and chronic toxic effects observed in other studies of vegetable oils and animal fats.

Spills of petroleum and vegetable oils and animal fats can affect drinking water supplies, and they have forced the closing of water treatment systems. Rancid smells, fouling of beaches, and destruction of recreational areas have been reported after spills of vegetable oils and animal fats.

Small spills of petroleum and vegetable oils and animal fats can cause significant environmental damage. Real-world examples of oil spills demonstrate that spills of petroleum oils and vegetable oils and animal fats do occur and produce deleterious environmental effects. In some cases, small spills of vegetable oils can produce more environmental harm than numerous larger spills of petroleum oils.

Because petroleum oils and vegetable oils and animal fats exhibit similar behavior in the environment, similar methods are used to contain them and attempt to clean them up after a spill. Because every spill is different, decisions on what cleanup methods are most effective and least harmful to the environment must be made case-by-case, considering the nature of the oil, the characteristics of the contaminated area, and the proximity of the spill to environmentally sensitive areas.

Once oil is spilled in the environment, however, the opportunities for reducing environmental damage and other adverse effects are limited. Although methods for rescuing and cleaning oil-contaminated birds, otters, and other wildlife have improved, only a small proportion of affected animals are recovered, and even fewer of the rescued animals survive. Further, by affecting current and future food sources, nesting habitats, and reproduction, oil spills can damage the environment long after the spilled oil has been removed from the environment. Prevention measures and rapid response offer the only effective means of minimizing the immediate, devastating effects and long-term environmental effects of spills of petroleum and non-petroleum oils, including vegetable oils and animal fats.

In summary, EPA finds that Petitioners' arguments about the manner in which environmental species die or become injured following spills of vegetable oils and animal fats, their claims about degradation of oil in the environment, and their assertion that fats are essential to humans and wildlife in no way obviate the need to prevent spills of vegetable oils and animal fats that can cause lasting environmental damage. Nor do the Petitioners' claims obviate the need to reduce environmental damage from these spills by planning in advance for effective response resources and actions. EPA hereby declines to modify the July 1, 1994, Final Rule.

Dated: October 1, 1997.

Timothy Fields, Jr.,

Acting Assistant Administrator, Office of Solid Waste and Emergency Response.

Acronym List

ACP—Area Contingency Plan
BOD—Biological Oxygen Demand
CFR—Code of Federal Regulations
COPs—Cholesterol Oxidation Products
CWA—Clean Water Act
DNA—Deoxyribonucleic Acid
DNR—Department of Natural Resources
DOT—Department of Transportation
EFA—Essential Fatty Acids
EPA—Environmental Protection Agency
ERNS—Emergency Response Notification System
FAO/WHO—Food and Agriculture Organization/World Health Organization
FR—Federal Register
FRP—Federal Response Plan
FWS—Fish and Wildlife Service
IARC—International Agency for Research on Cancer
Institute—Institute of Shortening and Edible Oils, Inc.
LC_{50}—Lethal Concentration 50
LD_{50}—Lethal Dose 50
LOPs—Lipid Oxidation Products
MOU—Memorandum of Understanding
NAS—National Academy of Sciences
NOAA—National Oceanic and Atmospheric Administration
NRC—Nuclear Regulatory Commission
NRC—National Response Center
OPA—Oil Pollution Act
PAHs—Polynuclear Aromatic Hydrocarbons
PCBs—Polychlorinated Biphenyls
PUFA—Polyunsaturated Fatty Acid (n-6 PUFA, including essential fatty acid linoleic acid; n-3 PUFA, including the essential fatty acid, a-linolenic acid)
RCRA—Resource Conservation and Recovery Act
RSPA—Research and Special Projects Administration
SPCC—Spill Prevention Countermeasure and Control
USDA—United States Department of Agriculture
USDHHS—United States Department of Health and Human Services
USDOC—United States Department of Commerce
USDOI—United States Department of Interior
USEPA—United States Environmental Protection Agency

Bibliography

Abel, P.D. (1996). *Water Pollution Biology.* Taylor and Francis, London, United Kingdom, 113–163.

Abernathy, Charles O., Robert Cantilli, Julie T. Du, and Orville A. Levander. (1992). Essentiality Versus Toxicity: Some Considerations in the Risk Assessment of Essential Trace Elements. In: J. Saxena, editor, *Hazard Assessment of Chemicals.* Hemisphere Publishing Corp., New York, NY, Volume 8.

Albers, P.H. (1977). Effects of External Applications of Fuel Oil on Hatchability of Mallard Eggs In: D.A. Wolfe, editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems.* Pergamon Press, Oxford, pp. 158–163.

Albers, P.H. (1995). Oil, Biological Communities and Contingency Planning In: L. Frink, K. Ball-Weir, and C. Smith, Editors. *Wildlife and Oil Planning. Response, Research, and Contingency Planning.* Tri-State Bird Rescue and Research, Newark, Delaware, pp. 1–9.

Alexander, Maurice M. (1983). Oil, Fish and Wildlife and Wetlands: A Review. *Northeastern Environmental Science* 2 (1): 13–24.

Allen, A. and W.G. Nelson. (1983). Canola Oil As a Substitute for Crude Oil in Cold Water Tests. *Spill Technology Newsletter,* January-February, pp. 4–10.

Amdur, Mary O., John Doull and Curtis D. Klaassen. (1991). *Casarett And Doull's Toxicology.* Pergamon Press, New York, NY, Fourth Edition, pp. 12–126, 565–680.

Andrews, J.S., W.H. Griffith, J.F. Mead, and R.A. Stein. (1960). Toxicity of Air-Oxidized Soybean Oil. *J. Nutrition* 70:199–210.

Aqua Survey, Inc. (1993). Diesel Fuel, Beef Tallow, RBD Soybean Oil and Crude Soybean Oil: Acute Effects on the Fathead Minnow, Pimephales Promelas. Study # 93–136, May 21, 1993.

Artman, N.R. (1969). The Chemical and Biological Properties of Heated and Oxidized Fats. In: R. Poaletti and D. Krichevsky, Editors, *Advances in Lipid Research,* pp. 245–330.

Berardi, L.C. and L.A. Goldblatt. (1980). Gossypol In: I.E. Liener, Editor, *Toxic Constituents of Plant Foodstuffs.* Second Edition, Academic Press, New York, pp. 183–237.

Boyd, E.M. (1973). *Toxicity of Pure Foods.* CRC Press, Cleveland, OH, pp. 71–111.

Brekke, O.L. (1980). Edible Oil Processing—Introduction In: American Soybean Association and American Oil Chemists' Society. *Handbook of Soy Oil Processing and Utilization.* St. Louis, MO and Champaign, IL, pp. 67–69.

Carroll, K.K. (1991) *Am. J. Clin. Nutr.* 53 (Suppl 4):1064S. Cited in Hui, Y.H. (1996a). *Bailey's Industrial Oil and Fat Products, Edible Oil and Fat Products: General Application.* John Wiley & Sons, Inc., New York, NY, Volume 1, Fifth Edition, pp. 194–196.

Chemical Hazards Response Information System (CHRIS). Department of Transportation, U.S. Coast Guard. January, 1991.

Chemical Hazards Response Information System (CHRIS). Department of Transportation, U.S. Coast Guard. 1995.

Clark, R.B. (1993). *Marine Pollution.* Clarendon Press, Oxford, 3rd Edition, Chapter 3, pp. 28–52.

Croxall, J.P. (1975). The Effect of Oil on Nature Conservation, Especially Birds. In: *Petroleum and the Continental Shelf of Northwest Europe.* v. 2: 93–101, Environmental Protection. Inst. Petrol., London. As cited in Alexander, 1983.

Croxall, J.P. (1977). The Effects of Oil on Seabirds. *Rapp. P-v. Reun. Cons. Int. Explor. Mer* 171:191–195, 1977 In: National Academy of Sciences. *Oil in the Sea.* Effects, pp. 430–436.

Crump-Wiesner, Hans J. and Allen L. Jennings. Properties and Effects of Nonpetroleum Oils. (1975). *Pro. of 1975 Conference on Prevention and Control of Pollution.* American Petroleum Institute, Washington, D.C., pp. 29–32.

Dubovkin, N.P., M.E. Tararyshkin, and L.D. Abashina. (1981). Crude Oil and Product Research: Vapor Pressure and Critical Parameters of Jet Fuel In: *Chemistry and Technology of Fuels and Oils.* Plenum Publishing Corporation, pp. 207–210. Translated from Russian; translated from Khimiya I Takhnologiya Topliv I Masel 17 (4):34–37, April, 1981, Consultants Bureau, New York.

Entrix, Inc. (1992). A Critical Review of Toxicity Values and an Evaluation of the Persistence of Petroleum Products for Use in Natural Resource Damage Assessments, Draft. Wilmington, DE. Prepared for American Petroleum Institute. December 18, 1992, pp. 108–120.

ENVIRON Corporation. (1993). Environmental Effects of Releases of Animal Fats and Vegetable Oils to Waterways. Arlington, VA June 3, 1993. Submitted by Petitioner.

Food and Agriculture Organization of the United Nations, World Health Organization (FAO/WHO). (1994). *Fats and Oils in Human Nutrition.* Expert Consultation, October 19–26, 1993, Rome. Published by Food and Agriculture Organization, Rome, pp. 1–102, 113–147.

Frankel, E.N. (1984). Lipid Oxidation: Mechanisms, Products and Biological Significance. *Journal of the American Oil Chemist Society* 61(12):1908–1917, December 1984.

Freedman. L.S., C. Clifford, and M. Messina. (1990). *Cancer Res.* 50:5710. In: Hui, Y.H. (1996a). *Bailey's Industrial Oil and Fat Products, Edible Oil and Fat Products: General Application.* John Wiley & Sons, Inc., New York, NY, Volume 1, Fifth Edition, pp. 194–196.

French, C.E., R.H. Ingram, J.A. Uram, G.P. Barron, and R.W. Swift. (1953). The Influence of Dietary Fat and Carbohydrate on Growth and Longevity in rats. *J. Nutr.* 51:329–339. Cited in: National Research Council, *Nutrient Requirement of Laboratory Animals.,* 4th Edition. National Academy Press, Washington, D.C., p. 20, 1995.

Frink, L. (1994). Statement on Regulatory Standards for the Transportation of Edible Oil. Tri-State Bird Rescue & Research, Inc., January 30, 1994.

Frink, L. and E.A. Miller. (1995). Principles of Oiled Bird Rehabilitation. *Wildlife and Oil Spills.* Tri-State Bird Rescue & Research, Inc., Newark, DE, pp. 61–68.

Gilman, A.G., L.S. Goodman, T.W. Rall, and F. Murad. (1985). *Goodman and Gilman's The Pharmacological Basis of Therapeutics.* Macmillan Publishing Company, Seventh Edition, pp. 1001–1003.

Goodrich, W.H. (1980). Environmental Concerns In: D.R. Erickson, E.H. Pryde, O.L. Brekke, T.L. Mounts, and R.A. Falb, Editors, *Handbook of Soy Oil Processing and Utilization.* American Soybean Association, St. Louis, MS and American Oil Chemists' Society, Champaign, IL, pp. 521–527.

Groenewold, J.C., R.F. Pico, K.S. Watson. (1982). Comparison of BOD Relationships for Typical Edible and Petroleum Oils. *Journal of the Water Pollution Control Federation* 54(4):398–405, April, 1982.

Hartung, R. (1965). Some Effects of Oiling on Reproduction of Ducks. *Journal of Wildlife Management* 29(4):872–874.

Hartung, R. (1967). Energy Metabolism in Oil-Covered Ducks. *Journal of Wildlife Management* 31:798–804.

Hartung, R. (1995). Assessment of the Potential for Long-Term Toxicological Effects of the Exxon Valdez Oil Spill on Birds and Mammals In: P.G. Wells, J.N. Butler, and J.S. Hughes, Editors, *Exxon Valdez Oil Spill: Fate and Effects in Alaskan Waters.* American Society for Testing and Materials, Philadelphia, PA, pp. 693–725.

Hayes, A. Wallace. (1982). *Principles and Methods Of Toxicology.* Raven Press, New York, NY, pp. 1–55.

Hayes, A. Wallace. (1989). Principles and Methods of Toxicology. Raven Press, New York, NY, Second Edition, pp. 75–76, 105–110, 169–220.

Hazardous Substances Data Base (HSDB), National Library of Medicine, 1997.

Hendricks, J.D., R.O. Sinnhuber, P.M. Loveland, N.E. Pawlowski, and J.E. Nixon. (1980a). Hepatocarcinogenicity of Glandless Cottonseeds and Refined Cottonseed Oil to Rainbow Trout (Salmo gairdneri). *Science* 208:309–310.

Hendricks, J.D., R.O. Sinnhuber, J.E. Nixon, J.H. Wales, M.S. Masri, and D.P.H. Hsieh. (1980b). Carcinogenic Response of Rainbow Trout (Salmo gairdneri) to Aflatoxin Q_1 and Synergistic Effect of Cyclopropenoid Fatty Acids. *JNCI* 64:523–528.

Hendricks, J.D., T.R. Meyers, and D.W. Shelton. (1984). Histological Progression of Hepatic Neoplasia in Rainbow Trout (Salmo gairdneri). In: K.L. Hoover, Editor, *Use of Small Fish Species in Carcinogenicity Testing.* National Cancer Institute Monograph 65, NIH Publication Number 84–2653, National Institutes of Health, Bethesda, Maryland, pp. 321–336, May, 1984.

Hoffmann, G. (1989). *The Chemistry and Technology of Edible Oils and Fats and their High Fat Products.* Academic Press, London, pp. 1–28, 343–363.

Hui, Y.H. (1996a). *Bailey's Industrial Oil and Fat Products, Edible Oil and Fat Products: General Application.* John Wiley & Sons, Inc., New York, NY, Volume 1, Fifth Edition, pp. 1–280, 397–439.

Hui, Y.H. (1996b). *Bailey's Industrial Oil and Fat Products, Edible Oil and Fat Products: Oils and Oilseeds.* John Wiley & Sons, Inc., New York, NY, Volume 2, Fifth Edition, pp. 1–689.

Hui, Y.H. (1996d). *Bailey's Industrial Oil and Fat Products, Edible Oil and Fat Products: Products and Application Technology.* John Wiley & Sons, Inc., New York, NY, Volume 4, Fifth Edition, pp. 1–655.

Institute of Shortening and Edible Oils, Inc. (1985). *Treatment of Wastewaters From Food Oil Processing Plants in Municipal Facilities.* October, 1985, pp. 1–18.

International Agency for Research on Cancer (IARC). (1984). *Evaluation of the Carcinogenic Risk of Chemicals to Humans: Polynuclear Aromatic Compounds, Part 2, Carbon Blacks, Mineral Oils and Some Nitroarenes.* IARC, France, Volume 33, pp. 29–31 and 87–168.

International Agency for Research on Cancer (IARC). (1989). *Evaluation of the Carcinogenic Risk of Chemicals to Humans: Occupational Exposures in Petroleum Refining: Crude Oil and Major Petroleum Fuels.* IARC, Lyon, France, Volume 45, pp. 13–272.

Kanazawa, A., S. Teshima, and K. Ono. (1979). Relationship Between Essential Fatty Acid Requirements of Aquatic Animals and the Capacity for Bioconversion of Linolenic Acid to Highly Unsaturated Fatty Acids. *Comp. Biochem. Physiol.* 63B:295–298.

Katan, M. B., P. L. Zock, and R.P. Mensink. (1995). Trans Fatty Acids and Their Effects on Lipoproteins in Humans. *Annual Review of Nutrition* 15:473–493.

Kiritsakis, A.K. (1991). *Olive Oil.* American Oil Chemists' Society, Champaign, Illinois, pp. 25–33, 104–127, and 157–161.

Klaassen, Curtis D., Mary O. Amdur and John Doull. (1986). *Casarett And Doull's*

Toxicology. Macmillan Publishing Company, New York, NY, Third Edition, pp. 11–98 and 519–635.

Kooyman, G.L., R.W. Davis, and M.A. Castellini. (1977) Thermal Conductance of Ememrsed Prinniped and Sea Otter Pelts Before and After Oiling with Prudhoe Bay Crude In: D.A. Wolfe, editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems*, Pergamon Press, Oxford, pp. 143–150.

Lawson, H. (1995). *Food Oils and Fats. Technology, Utilization, and Nutrition*. Chapman & Hall, New York, NY, pp. 3–27.

Lee, R.F. (1977). Accumulation and Turnover of Petroleum Hydrocarbons in Marine Organisms In: D.A. Wolfe, Editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms*, Pergamon Press, Oxford, pages 60–70.

Lee, D.J., J.H. Wales, J.L. Ayres, and R.O. Sinnhuber. (1968). Synergism between Cyclopropenoid Fatty Acids and Chemical Carcinogens in Rainbow Trout (Salmo gairdneri). *Cancer Research* 28:2312–2318, November, 1968.

Leighton, F.A. (1995). The Toxicity of Petroleum Oils to Birds: An Overview. In: L. Frink, K. Ball-Weir, And C. Smith, Editors, *Wildlife and Oil Spills: Response, Research, and Contingency Planning*, Tri-State Bird Rescue & Research, Newark, Delaware, pp. 10–22.

Levy, J.R., K.A. Faber, L. Ayyash, and C.L. Hughes. (1995). The Effect of Prenatal Exposure to the Phytoestrogen Genistein on Sexual Differentiation in Rats. Second International Conference on Phytoestrogens. October 17–20, 1993. Little Rock, Arkansas. Proceedings of the Society for Experimental Biology and Medicine 208 (1):60–66, January, 1995.

Lewis, A., P. S. Daling, T. Strom-Kristiansen and P. J. Brandvik. (1995). The Properties and Behavior of Crude Oil Spilled at Sea. *Pro. Second International Oil Spill Research and Development Forum*. International Maritime Organization, London, United Kingdom, Volume 1, May 1995, pp. 408–420.

Lewis, R.J. (1996). *Sax's Dangerous Properties of Industrial Materials*. Ninth Edition, Van Nostrand Reinhold, New York, Volume 1: pp. xiii-xxvii, Volume 2: pp. 922, 924, 1143–1144, Volume 3: pp. 2054, 2372–2373, 2514, 2561, 2573–2574, 2894, 3340, 3367.

List, G. R. and D. R. Erickson. (1980). Storage, Stabilization and Handling In: D.R. Erickson, E.H. Pryde, O.L. Brekke, T.L. Mounts, and R.A. Falb, Editors, *Handbook of Soy Oil Processing and Utilization*. American Soybean Association, St. Louis, MS and American Oil Chemists' Society, Champaign, IL, pp. 272–98 and 300–301.

Lyall, S. ''And Still the Birds Die, Out in the Poisoned Sea.'' New York Times International, April 26, 1996.

Material Safety Data Sheet (MSDS). (1997). Corn Oil. Fisher Scientific, Fairlawn, NJ.

Mattson, F. H. (1973). Potential Toxicity of Food Lipids. In: National Academy of Sciences, *Toxicants Occurring Naturally in Foods*. National Academy of Sciences Press, Washington, D.C., pp. 189–209.

McKelvey, R.W., I. Robertson and P.E. Whitehead. (1980). Effect of Non-Petroleum Oil Spills on Wintering Birds Near Vancouver. *Marine Pollution Bulletin* 11:169–171.

Mecklenburg, T.A., S.D. Rice, and J.F. Karinen. (1977). Molting and Survival of King Crab (Paralithodes ''Camtschatica'') and Coonstripe Shrimp (Pandalus Hypsinothus) Larvae Exposed to Cook Inlet Crude Oil Water-Soluble Fraction In: D.A. Wolfe, Editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystem*, Pergamon Press, Oxford, pp. 221–228.

Merck Index. (1989). 11th Edition. Merck and Co., Inc., Rahway, NJ. Pp. 2528, 2550, 5173, 5245, 6951, 8127–8128.

Metcalf and Eddy, Inc. (1972). *Wastewater Engineering: Collection, Treatment, Disposal*, Consulting Editors, V.T. Chow, R. Eliassen, and R.K. Linsley, McGraw Hill, Inc., New York, pp. 239–240.

Michael, A.D. (1977). The Effects of Petroleum Hydrocarbons on Marine Populations and Communities. In: D.A. Wolfe, Editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms*, Pergamon Press, Oxford, pages 129–137.

Miller, A.M., E.T. Sheehan, and M.G. Vavich. (1969). Prenatal and Postnatal Mortality of Offspring of Cyclopropenoid Fatty Acid-Fed Rats. *Proc. Soc. Exp. Biol. Med.* 131:61–66, 1969.

Minnesota Department of Conservation, Division of Game and Fish. Waterfowl Mortality Caused by Oil Pollution of the Minnesota and Mississippi Rivers in 1963. (1963). In *Proceedings of the 20th Annual Meeting of the Upper Mississippi River Conservation Committee*, pp. 149–177.

Mudge, S. M., M. A. Salgado and J. East. (1993). Preliminary Investigations into Sunflower Oil Contamination Following the Wreck of the M. V. Kimya. *Marine Pollution Bulletin* 26 (1): 40–43.

Mudge, S.M., H. Saunders and J. Latchford. (1994). Degradation of Vegetable Oils in the Marine Environment. *Countryside Commission for Wales Report*, CCW, Bangor, pp. 1–41.

Mudge, Stephen M. (1995). Deleterious Effects from Accidental Spillages of Vegetable Oils. *Spill Science and Technology Bulletin* 2 (2/3): 187–191.

Mudge, Stephen M., Ian D. Goodchild and Matthew Wheeler. (1995). Vegetable Oil Spills on Salt Marshes. *Chemistry and Ecology* 10: 127–135.

Mudge, Stephen M. (1997a) Presentation, *Third International Ocean Pollution Symposium*, April 6–11, 1997, Harbor Branch Oceanographic Institution, Ft. Pierce, Florida.

Mudge, Stephen. (1997b) Can Vegetable Oils Outlast Mineral Oils in the Marine Environment? *Marine Pollution Bulletin*, in press.

Murata, S., F. Tanaka, and J. Tokunaga. (1993). Measurement of Vapor Pressure of a Series of Edible Oils. *J. Fac. Agr., Kyushu Univ.* 38(1–2): 9–18.

Murray, M.W., J.W. Andrews, and H.L. DeLoach. (1977). Effects of Dietary Lipids, Dietary Protein and Environmental Temperatures on Growth, Feed Conversion and Body Composition of Channel Catfish. *J. Nutr.* 107: 272–280. Cited in: National Research Council, *Nutrient Requirements of Warmwater Fishes and Shellfishes*. National Academy Press, pp. 9–11, 1983.

National Academy of Sciences (NAS), National Research Council (NRC). (1977a). *Drinking Water and Health*. NAS, Washington, D.C., Volume 1, pp. 19–62, 252–253, and 265–270.

National Academy of Sciences (NAS), National Research Council (NRC). (1977b). *Nutrient Requirements of Rabbits*. National Academy Press, Washington, D.C., Second Revised Edition, pp. 2–28.

National Academy of Sciences (NAS), National Research Council (NRC). (1981a). *Nutrient Requirements of Coldwater Fishes*. National Academy Press, Washington, D.C., Number 16, pp. 6–53.

National Academy of Sciences (NAS), National Research Council (NRC). (1981b). *Nutrient Requirements of Goats: Angora, Dairy, and Meat Goats in Temperate and Tropical Countries*. National Academy Press, Washington, D.C., Number 15, pp. 2–5 and 74–76.

National Academy of Sciences (NAS), National Research Council (NRC). (1983). *Nutrient Requirements of Warmwater Fishes and Shellfishes*. National Academy Press, Washington, D.C., Revised Edition, pp. 2–58.

National Academy of Sciences (NAS), National Research Council (NRC). (1984). *Nutrient Requirements of Beef Cattle*. National Academy Press, Washington, D.C., 6th revised edition, pp. 2–6 and 35–36.

National Academy of Sciences (NAS). (1985c). *Oil in the Sea—Inputs, Fates and Effects*. Chemical and Biological Methods. National Academy Press, Washington D.C. pp. 89–269.

National Academy of Sciences (NAS). (1985d). *Oil in the Sea—Inputs, Fates and Effects*. Fates. National Academy Press, Washington D.C., pp. 270–368.

National Academy of Sciences (NAS). (1985e). *Oil in the Sea—Inputs, Fates and Effects*. Effects. National Academy Press, Washington, D.C., pp. 369–547.

National Academy of Sciences (NAS), National Research Council (NRC). (1985). *Nutrient Requirements of Sheep*. National Academy Press, Washington, D.C., Sixth Revised Edition, pp. 2–8 and 28.

National Academy of Sciences (NAS), National Research Council (NRC). (1988). *Nutrient Requirements of Swine*. National Academy Press, Washington, D.C., Ninth Revised Edition, pp. 2–8 and 63–66.

National Academy of Sciences (NAS), National Research Council (NRC). (1992). *Nutrient Requirements of Mink and Foxes*. National Academy Press, Washington, D.C., Number 7, Second Revised Edition, pp. 9 and 19.

National Academy of Sciences (NAS), National Research Council (NRC). (1994). *Nutrient Requirements of Poultry*. National Academy Press, Washington, D.C., Ninth Revised Edition, pp. 11–1320–23, 27, 36–37, 40–42, 45, 69, and 75–77.

National Academy of Sciences (NAS), National Research Council (NRC). (1995). *Nutrient Requirements of Laboratory*

Animals. National Academy Press, Washington, D.C., Fourth Revised Edition, pp. 17–21, 60–71, 84–85, 97–100, 106–107, 126–129, and 141.

Nelson, G.J. (1990). *Health Effects of Dietary Fatty Acids.* American Oil Chemists' Society, Champaign, Illinois, pp. 1–263.

Newman, G.G. and D.E. Pollock. (1973). Organic Pollution of the Marine Environment by Pelagic Fish Factories in the Western Cape. *South African Journal of Science* 69:27–29.

Oil Pollution Act (OPA) Public Law 101–380, 104 Stat. 484.

Percy FitzPatrick Institute of African Ornithology. (1974). Fish Oil Kills Sea Birds. *African Wildlife* 28(4):24–25.

Phelps, R.A., F.S. Shenstone, A.R. Kemmerer, and R.J. Evans. (1965). A Review of Cyclopropenoid Compounds: Biological Effects of Some Derivatives. *Poultry Science* 44:358–394.

Rabbani, P.I., H.Z. Alam, S.J. Chirtel, R. Duvall, R.C. Jackson, and G. Ruffin. (1997). Subchronic Toxicity of Menhaden Oil (MO) and MaxEPA in Male and Female Rats. (Abstract). *Fundamental and Applied Toxicology,* Supplement 36(1), Part 2:42–43, March, 1997. Presented at 36th Annual Meeting of Society of Toxicology, Cincinnati, Ohio, March 9–13, 1997.

Rand, G.M. and S.R. Petrocelli. (1985). *Fundamentals of Aquatic Toxicology: Methods and Applications.* Hemisphere Publishing Corporation, New York, NY, pp.1–109 and 124–163.

Ratledge, C. (1994). Biodegradation of Oils, Fats, and Fatty Acids. In: C. Ratledge, Editor, *Biochemistry of Microbial Degradation.* Kluwer Academic Publishers, The Netherlands, pp. 89–141.

Rechcigl, Jr., M. (1981). *CRC Handbook of Nutritional Requirements in a Functional Context: Hematopoiesis, Metabolic Function, and Resistance to Physical Stress.* CRC Press, Inc., Boca Raton, FL, Volume II, pp. 80–81, 148–51, 302–330, 342–43, 349, 357, 364–66, 381–82, 388, 390, 392, and 394–95.

Rechcigl, Jr., M. (1983). *CRC Handbook of Naturally Occurring Food Toxicants.* CRC Press, Inc., Boca Raton, FL, pp. 15–17, 21–30, 101–104, 226.

Rescorla, A.R. and F.L. Carnahan. (1936). *Ind. Eng. Chem.* 28:1212–1213. In A.E. Bailey, *Bailey's Industrial Oil and Fat Products,* p. 55, 1945.

Rigger, D. (1997). Edible Oils: Are They Really That Different? *Proceedings of the 1997 International Oil Spill Conference.* Sponsored by U.S. Coast Guard, U.S. Environmental Protection Agency, American Petroleum Institute, International Petroleum Industry Environmental Conservation Association, and International Maritime Organization. Fort Lauderdale, Florida. April 7–10, 1997, pp. 59–61.

Roine, P., E. Uksila, H. Teir, and J. Rapola. (1960). Histopathological Changes in Rats and Pigs Fed Rapeseed Oil. *Zeitschrift feur Erneahrungswissenschaft* 1:118–124.

Rozemeijer, M.J.C., K. Booij, C. Swennen and J.P. Boon. (1992). Harmful Effects of Floating Lipophilic Substances Discharged from Ships on the Plumage of Birds. Netherlands Institute for Sea Research (NIOZ), Den Burg, Netherlands, pp. 1–17, April, 1992. Attachment to Amendments and Interpretation of the BCH Code and the IBC Code, International Maritime Association, July 17, 1992 and Annex 3, Draft Marine Environment Protection Committee Circular on Harmful Effects on Birds from Contact with Animal and Fish Oils and Fats and Vegetable Oils (Floating Lipophylic Substances).

Russell, D.J. and B.A. Carlson. (1978). Edible-Oil Pollution on Fanning Island, *Pacific Science* 32(1):1–15, University Press of Hawaii.

Salanitro, J.P., P. Dorn, M. Huesemann, K.O. Moore, I.A. Rhodes, L.M.R. Jackson, T.E. Vipond, M.M. Western, and H.L. Wisniewski. (1997). Crude Oil Hydrocarbon Bioremediation and Soil Ecotoxicity Assessment. *Environ. Sci. Technol.* 31: 1769–1776.

Salgado, M. (1992). *Contamination of the Anglesey Coastline by Sunflower Oil from the Wreck of the M.V. Kimya.* Master of Science Thesis, School of Ocean Sciences, University College of North Wales, Menai Bridge, United Kingdom. April, 1992, pp. 1–107.

Salgado, M. (1995). *The Effects of Vegetable Oil Contamination on Mussels.* PhD Thesis, School of Ocean Sciences, University of Wales, Menai Bridge, Gwynedd, United Kingdom. October, 1995, pp. 1–219.

Sanders, H.L., J.F. Grassle, G.R. Hampson, L.S. Morse, S. Garner-Price and C.C. Jones. (1980). Anatomy of an Oil Spill: Long-term Effects from the Grounding of the Barge Florida Off West Falmouth, Massachusetts. *Journal of Marine Research* 38(2): 265–380.

Sellers, E.A. and D.G. Baker. (1960). Coronary Atherosclerosis in Rats Exposed to Cold. *Can. Med. Assoc. J.* 83:6–13 In: M. Rechcigl, Editor, *CRC Handbook of Nutritional Requirements in a Functional Context, vol. II, Hematopoiesis, Metabolic Function, and Resistance to Physical Stress,* CRC Press, Inc., Boca Raton, Florida, 1981, pp. 527–528, 540.

Shaw, D.G. (1977). Hydrocarbons in the Water Column. In: D.A. Wolfe, Editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms,* Pergamon Press, Oxford, pages 8–18.

Sheehan, D.M. (1995). Introduction: The Case for Expanded Phytoestrogen Research. Second International Conference on Phytoestrogens. October 17–20, 1993. Little Rock, Arkansas. Proceedings of the Society for Experimental Biology and Medicine 208(1):3–5, January, 1994.

Smith, D.W. and S.M. Herunter. (1989). Birds Affected by a Canola Oil Spill in Vancouver Harbour, February, 1989. *Spill Technology Newsletter,* pp. 3–5 October–December 1989.

Steinberg, D. (1971). Phytanic Acid Storage Disease (Refsum's Disease). In: J.B. Stanbury, J.B. Wyngaarden, and D.S. Fredrickson, Editors, *Metabolic Basis of Inherited Disease.* 3rd Edition, McGraw-Hill, New York. Cited in Mattson, F.H. Potential Toxicity of Food Lipids, National Academy of Sciences, *Toxicants Occurring Naturally in Foods.* National Academy of Sciences Press, Washington, D.C., pp. 189–209, 1973.

Stickney, R.R. and J.W. Andrews. (1971). Combined Effects of Dietary Lipids and Environmental Temperature on Growth, Metabolism and Body Composition of Channel Catfish (Ictalurus punctatus). *J. Nutr.* 101:1703–1710. Cited in: National Research Council, *Nutrient Requirements of Warmwater Fishes and Shellfishes.* National Academy Press, Washington, D.C., pp. 9–11.

Stickney, R.R. and J.W. Andrews. (1972). Effects of Dietary Lipids on Growth, Food Conversion, Lipid and Fatty Acid Composition of Channel Catfish. *J. Nutr.* 102:249–258. Cited in: National Research Council, *Nutrient Requirements of Warmwater Fishes and Shellfishes.* National Academy Press, Washington, D.C., pp. 9–11.

Straughan, D. (1977). Biological Survey of Intertidal Areas in the Straits of Magellen in January, 1975, Five Months After the Metula Oil Spill In: D.A. Wolfe, Editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems,* Pergamon Press, Oxford, pp. 247–260.

Szaro, R.C. and P.H. Albers. (1977). Effects of External Applications of No. 2 Fuel Oil on Common Eider Eggs In: D.A. Wolfe, Editor, *Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems.* Pergamon Press, Oxford, pp. 164–167, 1977.

Takeuchi, T. and T. Watanabe. (1979). Studies on Nutritive Value of Dietary Lipids in Fish. XIX. Effect of Excessive Amounts of Essential Fatty Acids on Growth of Rainbow Trout. *Bull. Jpn. Soc. Sci. Fish.* 45:1517–1519. Cited in: National Research Council, *Nutrient Requirements of Warmwater Fishes and Shellfishes.* National Academy Press, Washington, D.C., pp. 9–11.

Tannenbaum, A. (1942). *Cancer Res.* 2:468. Cited in: Hui, Y.H. (1996a). *Bailey's Industrial Oil and Fat Products, Edible Oil and Fat Products: General Application.* John Wiley & Sons, Inc., New York, NY, Volume 1, Fifth Edition, pp. 194–196.

Teal, J.M. (1977). Food Chain Transfer of Hydrocarbons, pp. 71–77 In: Wolfe, D.A. *Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms.* Pergamon Press, New York, NY, Chapters 8, 15, 16 and 23.

Ullrey, D.E. (1996). Personal Communication to Barbara Davis on Nutritional Ecology of the Ruminant. June 18, 1996.

U.S. Department of Commerce (USDOC), National Oceanic and Atmospheric Administration (NOAA). (1992a). *An Introduction to Oil Spill Physical and Chemical Processes and Information Management.* April, 1992, Chapters 2 and 3, pp. 2–1 to 2–56 and 3–1 to 3–97.

U.S. Department of Commerce (USDOC), National Oceanic and Atmospheric Administration (NOAA). (1992b). *Shoreline Countermeasures Manual. Temperate Coastal Environments.* pp. 9–23 and 37–62, December, 1992.

U.S. Department of Commerce (USDOC), National Oceanic and Atmospheric Administration (NOAA). Memorandum of Record, June 3, 1993, from NOAA Hazardous Materials Response and Assessment Division, June 3, 1993.

U.S. Department of Commerce (USDOC), National Oceanic and Atmospheric Administration (NOAA). (1994). *Options for Minimizing Environmental Impacts of*

Freshwater Spill Response. NOAA and American Petroleum Institute, September 1994, pp. 2–11, 122–124.

U.S. Department of Commerce (USDOC), National Oceanic and Atmospheric Administration (NOAA). (1996). Damage Assessment and Restoration Program. *Injury Assessment: Guidance Document for Natural Resource Damage Assessment Under the Oil Pollution Act of 1990.* Silver Spring, Maryland, Appendix C, Oil Behavior, Pathways, and Exposure, pps. C–1–24 and Appendix D, Adverse Effects From Oil, pps. D–1–69, August, 1996.

U.S. Department of Health & Human Services (USDHHS), Public Health Service (PHS), 1963. *Report on Oil Spills Affecting the Minnesota and Mississippi Rivers, Winter of 1962–1963.* Cincinnati, Ohio, pp. 1–40.

U.S. Department of Health & Human Services (USDHHS). (1990). *Healthy People 2000.* Conference Edition, pp. 119–137 and 390–436.

U.S. Department of Health & Human Services (USDHHS). Agency for Toxic Substances and Disease Registry (ATSDR). (1995a). *Toxicological Profile for Gasoline,* pp. 9–111, June, 1995.

U.S. Department of Health & Human Services (USDHHS). Agency for Toxic Substances and Disease Registry (ATSDR). (1995b). *Toxicological Profile for Fuel Oils.* pp. 9–109, June 1995.

U.S. Department of Health & Human Services (USDHHS). Agency for Toxic Substances and Disease Registry (ATSDR). (1995c). *Toxicological Profile for Jet Fuels (JP4 and JP7).* pp. 7–74, June 1995.

U.S. Department of the Interior (USDOI), Fish & Wildlife Service (FWS). (1994). Submitted to U.S. Environmental Protection Agency, 1994. FWS Memorandum from Peter H. Albers, Leader, Contaminant Ecology Group to Oil Spill Response Coordinator, December 29, 1993. U.S. Department of the Interior (USDOI), Fish & Wildlife Service (FWS). U.S. Fish and Wildlife Service Letter from Michael J. Spear, Assistant Director, Ecological Services, to Ms. Ana Sol Gutierrez, Research and Special Projects Administration, U.S. Department of Transportation, April 11, 1994.

U.S. Department of Transportation (USDOT), Coast Guard (CG). (1996). *Response Plans for Marine Transportation-Related Facilities: Final Rule.* 61 FR 7890, 7907–7908, Feb. 29, 1996.

U.S. Environmental Protection Agency (USEPA), Memorandum of Understanding between the Secretary of Transportation and the EPA Administrator, dated November 24, 1971 (36 FR 24080).

U.S. Environmental Protection Agency (USEPA), Office of Research and Development (ORD). (1976). *Acute Toxicity of Selected Organic Compounds to Fathead Minnows.* EPA–600/3–76–097, Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Duluth, Minnesota, October, 1976, pp 1–13.

U.S. Environmental Protection Agency (USEPA). (1978). *Identification of Conventional Pollutants.* 43 FR 32857–32859, July 28, 1978.

U.S. Environmental Protection Agency (USEPA). (1979). *Identification of Conventional Pollutants, Final Rule.* 44 FR 44501–44503, July 30, 1979.

U.S. Environmental Protection Agency (USEPA). (1980). *Water Quality Criteria Documents; Availability.* 45 FR 79318–79379, November 28, 1980.

U.S. Environmental Protection Agency (USEPA), Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response (OSWER). (1988). *The Oil Spill Prevention, Control, and Countermeasures Program Task Force Report.* May 13, 1988.

U.S. Environmental Protection Agency. (1994a). Memorandum from Kathleen Bogan, ABB to Dana Stalcup, Oil Program Branch, USEPA, June 22, 1994.

U.S. Environmental Protection Agency (USEPA, 1994b). *Oil Pollution Prevention; Non-Transportation-Related Onshore Facilities; Final Rule.* 59 FR 34070, 3408, Appendix E, July 1, 1994.

U.S. Environmental Protection Agency (USEPA, 1994c). *Request for Data and Comment on Response Strategies for Facilities that Handle, Store, or Transport Certain Non-Petroleum Oils.* 59 FR 53742, 53743, October 26, 1994, p. 5.

U.S. Environmental Protection Agency (USEPA), Office of Solid Waste and Emergency Response (OSWER), Environmental Response Team. (1995). Hazardous Material Incident Response Training Program, Edison, NJ, 1995 Review, Section 4, pp. 1–27.

U.S. Environmental Protection Agency (USEPA), Office of Solid Waste and Emergency Response (OSWER). Emergency Response Notification System (ERNS) database, 1996 and ''An Overview of ERNS,'' March, 1995.

Van Soest, P.J., *Nutritional Ecology of the Ruminant.* (1994). Comstock Publishing Associates, Ithaca, pp. 1–3, 7–11, 325–336.

Venosa, A.D., M.T. Suidan, B.A. Wrenn, K.L. Strohmeier, J.R. Haines, B.L. Eberhart, D. King, and E. Holder. (1996). Bioremediation of an Experimental Oil Spill on the Shoreline of Delaware Bay. *Environ. Sci. Technol.* 30:1764–1775.

Weiss, T.J. (1983). *Food Oils and Their Use.* Second Edition, Avi Publishing Co., Inc., Westport, CT, pp. 10–13.

Whiticar, S., M. Bobra, M. Fingas, P. Jokuty, P. Liuzzo, S. Callaghan, F. Ackerman and J. Cao. (1993). *A Catalogue of Crude Oil and Oil Product Properties.* Environment Canada, Ottawa, Ontario, 1992 Edition, February 1993, pp. 111–119, 154–156, 160–165, 215–266.

Williams, T.M., J. McBain, R.K. Wilson, and R.W. Davis. (1990). Clinical Evaluation and Cleaning of Sea Otters Affected by the T/V Exxon Valdez Oil Spill. *Sea Otter Symposium,* U.S. Department of the Interior, Biological Report 90 (12), December, 1990, Washington, D.C., pp. 236–257.

Wisconsin, Department of Natural Resources. (1991a). Spill Cleanup from the Central Storage and Warehouse Fire on Cottage Grove Road in Madison. Memo of J.W. Brusca to K.R. Williams, May 14, 1991; Central Storage Warehouse Fire/Spill Cleanup.

Wisconsin, Department of Natural Resources. (1991b). Memo from T.J. Amman to Floyd Stutz, about May 31, 1991; Update on Clean Up Actions following the Fire in Early May, 1991.

Wisconsin, Department of Natural Resources. (1991c). Letter from T.J. Amman to T. Fitzgerald, Central Storage and Warehouse Co., Inc., August 1, 1991.

Wisconsin State Journal (1991a). *Walking in Wet Concrete.* May 5, 1991.

Wisconsin State Journal (1991b). Mike Flaherty. *Grease Fire Burning On.* May 6, 1991.

Wisconsin State Journal (1991c). Mike Flaherty. *Blaze Also Poses Environmental Threat.* May 6, 1991.

Wisconsin State Journal (1991d). Jonnel LiCari. *Rain Adds to Cleanup Woes.* May 6, 1991.

Wisconsin State Journal (1991e). Mike Flaherty. *Fire Assessment Begins.* May 9, 1991.

Wolfe, D.A. (1986). Source of Organic Contaminants in the Marine Environment: Ocean Disposal and Accidental Spills In: C.S. Giam and H.J.M. Dou, Editors, *Strategies and Advanced Techniques for Marine Pollution Studies: Mediterranean Sea.* Springer-Verlag, Berlin, pp. 237-288.

Yannai, S. (1980). Toxic Factors Induced by Processing In I.E. Liener, Editor, *Toxic Constituents of Plant Foodstuffs,* Academic Press, New York, Second Edition, pp. 371–427.

Zoun, P.E.F., A.J. Baars and R.S. Boshuizen. (1991). A Case of Seabird Mortality in the Netherlands Caused by Spillage of Nonylphenol and Vegetable Oils, Winter 1988/1989. *Sula* 5 (3): 101–103. Summary of a report, published in Dutch.

Appendix I—Supporting Tables

TABLE 1.—COMPARISON OF PHYSICAL PROPERTIES OF VEGETABLE OILS AND ANIMAL FATS WITH PETROLEUM OILS

Oil type	Solidification point	Solubility	Specific Gravity at 25°C unless otherwise specified	Vapor pressure (mmHg)
Edible Oils				
Tallow	40 to 46°C [1]	Insoluble in water [1]	0.87 at 80°C [3]	
Corn oil	14 to 20°C [4]	Insoluble in water; soluble in acetone.[1,2]	0.916–0.921 [4], 0.91875.[5]	Negligible.[6]
Coconut oil	Solid to liquid at 15°C, 1 atm.[7]	Insoluble in water; very soluble in ether.[1]	0.922 [7]	
Rapeseed/Canola oil	−2 to −10°C; liquid at 15°C.[4]	Insoluble in water; soluble in chloroform and ether.[4]	0.913–0.917 [8]	250°C, 0.535mmHg.[9]
Fish oil	−2 to 4°C; liquid at 15°C.[4]	Insoluble in water [1]	0.93 at 20°C.[7]	
Soybean oil	−10 to −16°C; liquid at 15°C.[5]	Insoluble in water and acetone.[1]	0.916–0.922 [4], 0.9175 [5]	250°C, 0.351mmHg.[9]
Cottonseed oil	0 to −5°C; liquid at 15°C.[4]	Insoluble in water; slightly soluble in alcohol.[1]	0.915–0.921 [4], 0.917 [5]	250°C, 0.317mmHg.[9]
Palm oil	Solid to liquid at 15°C, 1 atm.[7]	Insoluble in water.[1]	0.920–0.927 (fruit), 0.952 (seed).[4]	
Lard	−2 to 4°C [1]	Insoluble in water or cold alcohol; soluble in ether and benzene.[1]	0.917 [4] <1 [1]	
Petroleum Oils				
Diesel	Liquid at 15°C, 1 atm [7]	Insoluble in water [7]	0.841 at 16°C [7]	38°C, 0.201mmHg.[9]
Fuel Oil #1 (kerosene)	Liquid at 15°C, 1 atm [7]	Insoluble in water; miscible with other petroleum solvents.[1]	0.80 [4]	21°C, 2.12–26.4mmHg.[11]
Fuel Oil 2–D	Liquid at 15°C, 1 atm [7]	Insoluble in water [7]	0.87–0.9 at 20°C [7]	21°C, 2.12–26.4mmHg.[11]
Crude	Liquid at 15°C, 1 atm [7]	Insoluble in water [7]	0.89 [8]	37.8°C, 3.27mmHg.[10]
Fuel Oil #6 Residual	Liquid at 15°C, 1 atm [7]	Insoluble in water [7]	0.95 approx. at 20°C [7]	37.8°C, 0.092mmHg.[10]
Jet Fuel JP #7				260°C, 2,480 mmHg.[12]
T 1				180–380°C, 6,907mmHg.[13]
T 6				170–450°C, 7,120mmHg.[13]

Oil type	Viscosity dynamic (centipoises)	Viscosity kinematic (centistokes)
Edible Oils		
Tallow	16.5 at 100°C [3]	
Corn oil	30.8 at 40°C [5]	
Coconut oil	32.6 at 32°C [7]	29.79 at 37.8°C.[14]
Rapeseed/Canola oil		50.64 at 37.8°C [14], 62.6 at 25°C, 36.7 at 40°C for RBD Soybean Oil.[5]
Fish oil		32.7 at 37.8°C (cod liver 12).[14]
Soybean oil	28 at 40°C [15]	28.49 at 37.8°C [14], 50.1 at 25°C, 28.9 at 40°C.[5]
Cottonseed oil	34 at 40°C [15]	38.88 at 37.8°C.[14]
Palm oil		
Lard	45 at 40°C [15]	44.41 at 37.8°C.[14]
Petroleum Oils		
Diesel	11.9 at 37.8°C [7]	6.8 at 20°C.[10]
Fuel Oil #1 (kerosene)	1.15 at 21°C [7]	1.7 at 15°C.[10]
Fuel Oil 2–D	1.97 at 21°C [7]	2.0 to 3.6 at 38°C.[10]
Crude	5.5 at 21°C [7]	5.96 at 20°C.[10]
Fuel Oil 6 Residual	123 to 233 at 20°C [10]	>130 at 40°C.[10]

[1] HSDB: Hazardous Substances Data Base. National Library of Medicine, 1997.
[2] USDOC/NOAA, 1994.
[3] Chemical Hazards Response Information System (CHRIS), DOT, USCG, January, 1991.
[4] Merck Index, 1989.
[5] Hui, 1996a, 1996b.
[6] Material Safety Data Sheet (MSDS), 1997, Corn Oil, Fisher Scientific.
[7] Chemical Hazards Response Information System (CHRIS), Department of Transportation, U.S. Coast Guard, 1995.

[8] Allen and Nelson, 1983.
[9] Murata et al., 1993.
[10] Whiticar et al., 1993.
[11] U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, 1995b.
[12] U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, 1995c.
[13] Dubovkin et al.,1981. Translated.
[14] Rescorla and Carnahan, 1945.
[15] Weiss, 1983.

TABLE 2.—COMPARISON OF VEGETABLE OILS AND ANIMAL FATS WITH PETROLEUM OILS

	Vegetable oil/animal fats	Petroleum oils
Chemical Properties:		
Chemical Structure	Triglycerides (triacylglycerols), cholesterol, phospho lipids, fatty acids, other components in crude oils.[1,2,3].	Alkanes, cycloalkanes, aromatic hydrocarbons, polynuclear aromatic hydrocarbons (PAHs), other components in crude oils.[4]
Chemical Form	Some liquids, some solids.[1,5,6,7,8,9]	Some liquids, some solids.[10,11,12,13]
Physical Properties:		
Density	Most 0.908–0.927 at 20 ° C; most float on water, some sink.[1,5,6,7,9,14].	Most 0.80–0.95 at 20° C; most float on water, some sink.[8,9,14]
Solubility	Most insoluble in water, soluble in organic solvents.[6,8,9].	Most insoluble in water, soluble in organic solvents.[6,8, 12]
Viscosity	Wide range, depends on temperature.[1,5,7,8,15,16].	Wide range, depends on temperature.[8,10]
Volatility	Generally small proportion volatile, most not volatile.[1,5,13,17].	Some fractions (e.g., gasoline) volatile, some not volatile; 11–90% volatile, depending on type of oil.[10,11,12,18]
Environmental Fate:		
Environmental Distribution	Oil found in water, soil/sediment, biota; usually little in air.[1,5,19,20,21,22,23].	Oil found in water, air, soil/sediment, biota.[4,12,24,25,26,27,28,29,30,31,32,33]
Persistence	May persist in environment for many years or degrade rapidly; depends on oil, media, environmental conditions where spilled.[22,34,35,36,37].	May persist in environment for many years; depends on oil, media, environmental conditions where spilled.[6, 30,38,39]
Chemical, Physical, and Biological Reactions.	Oxidation, hydrolysis, polymerization, photolysis, other chemical reactions; degraded by microorganisms, metabolized by plants and animals.[1,2,3,40,41].	Oxidation, photolysis, weathering processes; degraded by microorganisms; petroleum components taken up by plants and animals, metabolized by macroinvertebrates and some other animals.[4,30,33]
Toxic Components, Degradation Products	Some oils contain toxic components or may be degraded to form toxic products.[1,2,43,44,45].	Many contain benzene, PAHs, and other toxic components; may be degraded to form toxic products.[46,47,48]
Physical Effects:		
Smothering	Yes; suffocation when oil blocks aeration at water surface or depletes oxygen through biodegradation.[20,22,49,50,51,52,53].	Yes; suffocation from oxygen depletion.[30,47]
Coating	Yes, can cause hypothermia, increased need for food, loss of buoyancy, decreased ability to escape predators.[22,29,36,37,54,55,56,57,58,59].	Yes, can cause hypothermia, increased need for food, loss of buoyancy, decreased ability to escape predators.[28,29,47,54,55,56,57,58]
Egg Contamination	Yes; can be transferred from coated parents and kill embryos by blocking air exchange at egg surface.[22,29,54,55,56,57,58].	Yes; can be transferred from coated parents and kill embryos by blocking air exchange at egg surface and by toxicitytion.[28,29,47,56,57,60,61,62,63]
Food and Habitat Destruction	Yes; can cause starvation or ingestion of oiled food, destruction of future food sources, destruction of habitat, community effects.[22,29,55,56,57].	Yes, can cause starvation or ingestion of oiled food that clogs organs, destruction of future food sources, destruction of habitat, community effects.[28,29,47,54,55,56,57,58,61,64,65]
Lethality (LD_{50}, LC_{50})	Results vary by test, organism, conditionsG546,47,66,67 Tests submitted by Petitioners Other tests: Corn oil and cottonseed more lethal than mineral oil in albino rats—55 g/kg was LD50 for 5 days for corn oil and for 4 days for cottonseed oil; no fatalities at 130 g/kg with mineral oil for 15 days.[69] Other tests: Several free fatty acids intermediate in lethality in series of chemicals in fathead minnows.[70] Other tests: Mussels died after two weeks or more of exposure to low levels of oils (0.3 ml/min flowrate for oils, 300 ml/min flowrate seawater).[19,21].	Results vary by test, organism, conditions.[46,47,66,67,68] Tests submitted by petitioner Other tests: 0.5–28 ppm 96-hour LC50 static tests for some aromatic hydrocarbons for selected marine macroinvertebrates and fish.[46,47,68]
Acute Toxicity	Laxative, diarrhea, lipid pneumonia, decreased ability to escape predators; some vegetable oils, such as safflower oil, are irritating to human skin and eyes.[55,56,57,71,72]	Laxative, decreased ability to escape predators, pneumonia; affects lung, liver, kidney, blood, gastrointestinal and nervous systems.[28,29,47,57]
Chronic Toxicity:		

TABLE 2.—COMPARISON OF VEGETABLE OILS AND ANIMAL FATS WITH PETROLEUM OILS—Continued

	Vegetable oil/animal fats	Petroleum oils
Cancer	High-fat diets and diets containing certain types of fats increase cancer incidence in studies of laboratory animals and epidemiological studies.[1,73,74,75,76,77,78].	Benzene and some PAHs are human carcinogens; certain crude oil fractions and petroleum products sufficient evidence of carcinogenicity in laboratory animals and associated with increased cancer in refinery workers.[47,48,79]
Effects on Growth	High levels of some types of fats increase growth and obesity but early death and decreased reproductive ability in several species of animals; elevated levels of some oils or components decrease growth in some fish; growth inhibition in mussels exposed to low levels of sunflower oil.[1,21,35,74,78,80,81,82,83,84,85,86].	Petroleum hydrocarbons affect nearly all aspects of physiology and metabolism; reduced feeding rates in most animal species studied at concentrations similar to those in spills; benthic organisms especially sensitive; varying responses in marine plants.[28,29,38,47]
Reproductive and Developmental Effects	Decreased reproduction or growth and survival of offspring in some animals ingesting high levels of oils; kills embryos in eggs by physical effects, unknown whether toxicity also occurs.[22,55,56,57,74].	Affect broad range of reproductive and developmental processes; sensitivities to hydrocarbons vary widely between species and life stages; significant reproductive impairment rarely seen in field although coral, mussels, fiddler crabs,fish, birds, crustaceans, teleosts can be affected, some for years; decreased reproductive capacity and malformations in fish, birds; reduced egg production and toxicity in several bird species.[28,29,30,38,47,59,60,61,62]
Other Toxic Effects	Effects on shells of mussels exposed to low levels of oils, decreased foot extension activity; human and some animal studies show correlation of high levels of dietary fats with coronary artery disease, some types of cancer, hypertension, diabetes, obesity, altered immunity, altered steroid excretion, effects on bone modeling; increased atherosclerosis in rats fed high cholesterol levels; decreased lifespan in some animals consuming high levels of certain types of oils that increased growth and obesity.[1,21,35,73,74,78,86,87].	Affect broad range of organ systems and functions; increased vulnerability to disease and decreased growth and reproductive success; adverse skin effects in workers; components affect immune and hematopoeitic systems.[28,29,30,38,39,47,48]
Toxicity of Components or Degradation Products.	Most common chronic toxic effects of gossypol, a cottonseed oil component, in animals are cardiac irregularity, circulatory failure or rupture of red blood cells, and death; erucic acid in rapeseed oil and mustardseed oil causes cardiac effects, fat deposition in hearts of animals, growth suppression, anemia, and other effects, affects essential fatty acids; cyclopropene fatty acids in cottonseed and other oils suppress growth and impair female reproduction in laboratory animals, produce embryomortality in hens and rats, increase liver toxicity of other chemicals, and cause liver cancer in rainbow trout; oxidation products of animal fats and vegetable oils—cholesterol oxidation products can adversely affect the heart, immune system, and metabolism, and some lipid oxidation products may act in cancer development and affect atherosclerosis.[1,42,43,44,88,89,90,91,92,93].	Single exposures to benzene, a component of petroleum oils, at very high concentrations fatal in man; can cause central nervous system stimulation followed by depression and respiratory failure; can produce nausea, giddiness, headache, unconsciousness, convulsions, and paralysis; chronic exposure of humans to benzene can produce anemia and other blood effects and decrease immune defense mechanisms; some PAHs, components of petroleum oils, have reproductive effects and cause birth defects in animals and can affect skin, body fluids, and the immune system after short and long-term exposures in animals, and cause some respiratory effects in workers; some breakdown products are mutagenic or linked to carcinogenicity.[12,28,29,38,47,48,66,79,94]
Indirect Effects	High levels of oils upset fermentation and digestion in ruminants.[95].	Fuel oil no. 5 reduced herring population by decreasing amphipod grazers that control fungal damage to fish eggs.[47]
Aesthetics (Fouling, Rancidity)	Rancid odors of breakdown products; fouling of beaches, polymers formed in water and on sediments and concrete-like aggregates of oil and sand foul beaches.[1,2,3,5,19,21,22,34,35,96].	Fouling of beaches with tar balls and weathered oil.[31,32,33,47]
Fire/Explosion Hazard	Usually not a hazard, unless hexane or other chemicals present.[1,2,15,17].	Many petroleum products contain volatile chemicals that are flammable or explosive under certain conditions.[11,12,18,31,39]

TABLE 2.—COMPARISON OF VEGETABLE OILS AND ANIMAL FATS WITH PETROLEUM OILS—Continued

	Vegetable oil/animal fats	Petroleum oils
Interference With Water Treatment	Large amounts can overwhelm microorganisms used in water treatment plants; treatment plants must be shut down and alternative water supply provided to prevent disruption from spills.[96,97,98,99,100].	Spills can interfere with water treatment processes, requiring shutdown of plants and provision of alternate water supply; can contaminate groundwater.[30,52,97,98,99]

[1] Hui, 1996a
[2] Hoffmann, 1989
[3] Lawson, 1995a
[4] NAS, 1985a
[5] Hui, 1996b
[6] Hazardous Substances Data Base, National Library of Medicine, 1997
[7] CHRIS (Chemical Hazards Response Information System), DOT, 1991
[8] CHRIS (Chemical Hazards Response Information System), DOT, 1995
[9] Merck Index, 1989
[10] Whiticar et al., 1993
[11] Dubovkin et al., 1995
[12] USDHHS/ATSDR, 1995b
[13] Material Safety Data Sheet on Corn Oil, 1997
[14] Allen and Nelson, 1983
[15] Rescorla and Carnahan, 1936
[16] Weiss, 1983
[17] Murata et al., 1993
[18] USDHHS/ATSDR,1995a
[19] Salgado, 1992
[20] Mudge et al., 1993
[21] Mudge, 1995
[22] Crump-Wiesner and Jennings, 1975
[23] Russell and Carlson, 1978
[24] Sanders et al., 1980
[25] Shaw, 1977
[26] Lee, 1977
[27] Teal, 1977
[28] Alexander, 1983
[29] Hartung, 1995
[30] USDOC/NOAA, 1996
[31] USDOC/NOAA, 1992b
[32] Clark, 1993
[33] NAS, 1985d
[34] Mudge, 1997a
[35] Mudge, 1997b
[36] Minnesota, 1963
[37] USDHHS, 1963
[38] Entrix, 1992
[39] USDOC/NOAA, 1992a
[40] Hui, 1996d
[41] Ratledge, 1994
[42] Hayes, 1982
[43] Mattson, 1973
[44] Berardi and Goldblatt, 1980
[45] Rechcigl, 1983
[46] NAS, 1985c
[47] NAS, 1985e
[48] IARC, 1989
[49] Mudge et al., 1995
[50] Mudge et al., 1997b
[51] Straughan , 1977
[52] Groenewold et al., 1982
[53] Institute, 1985
[54] Michael, 1977
[55] USDOI/FWS, 1994
[56] Frink, 1994
[57] Frink and Miller, 1995
[58] Rozemeijer et al., 1992
[59] Smith and Herunter, 1989
[60] Albers, 1995
[61] Leighton, 1995
[62] Albers, 1977
[63] Szaro and Albers, 1977
[64] Croxall, 1975
[65] Lyall, 1996
[66] Klaassen et al., 1986
[67] Rand, 1985
[68] Mecklenburg et al., 1977
[69] Boyd, 1973
[70] USEPA, 1976
[71] Gilman et al., 1985
[72] Lewis, 1996
[73] USDHHS, 1990
[74] NAS/NRC, 1995

[75] Tannenbaum, 1942
[76] Carroll, 1990
[77] Freedman, 1990
[78] FAO/WHO, 1994
[79] IARC, 1984
[80] NAS/NRC, 1983
[81] NAS/NRC, 1981a
[82] Takeuchi and Watanabe, 1979
[83] Stickney and Andrews, 1971
[84] Stickney and Andrews, 1972
[85] Murray et al., 1977
[86] Salgado, 1995
[87] Sellers and Baker, 1960
[88] Frankel, 1984
[89] Hendricks et al., 1980a
[90] Phelps et al., 1965
[91] Miller et al., 1969
[92] Roine et al., 1960
[93] Yannai, 1980
[94] USDHHS/ATSDR, 1995d
[95] Van Soest, 1994
[96] Rigger, 1997
[97] USEPA, 1978; Identification of Conventional Pollutants, 43 FR 32857–32859, July 28, 1978
[98] USEPA, 1979; Final Rule, Identification of Conventional Pollutants, 44 FR 44501–44503, July 30, 1979
[99] Metcalf and Eddy, 1972
[100] Goodrich, 1980

TABLE 3. COMPARISON OF AQUA METHODS AND STANDARD ACUTE AQUATIC TESTING METHODS

Method	Number of species	Fish size	Acclimation
AQUA Report 1993	1—Fathead minnow	0.066±0.041 g, 20.4±3.7 mm, approximately 4 weeks old.	5 days.
USEPA/OPP 1982 (update 1985) [1].	2—1 warmwater, 1 coldwater (2—1 warmwater, 1 coldwater).	0.5-5 g, very young not used, longest no more than twice shortest (0.5-5g).	(At least 2 weeks).
ASTM 1986	List of recommended species.	0.5-5 usually, not very young, similar size and age, length of longest no more than twice shortest.	2 days or more with 100% dilution water and maximum temperature, change no more than 3 °C over 72 hours.
USEPA/OTS 1985 (update 1987).	Fathead minnow or other listed species.	2±1 cm recommended length	Held 12 to 15 days before testing; maintained in water of quality to be used in test at least 7 days.
USEPA/ORD 1985 (update 1991) {update 1993b} [2].	Species depends on regulatory requirements.	Age: 1–90 days {Age: 1–14 days}	At least 24 hours in 100% dilution water at temperature range of test.
APHA 1989	List; sensitive to effluent, material, envi. conditions.	Most sensitive life stage, depending on test purpose; longest no more than 1.5 times length of shortest.	Acclimate fish to lab conditions at least 14 days; 100% dilution water for at least 2 days.
OECD 1984	1 or more	Recommended total length for several species; 2±1 cm for fathead minnow; rationale if others.	12 days or more; fish exposed to water of test quality and temperature at least 7 days.
EEC 1984	1 or more	Recommended length 5±2 cm for fathead minnow.	12 days or more; fish exposed to water of test quality and temperature at least 7 days.

Method	Static test duration	Aeration
AQUA Report 1993	48 hours	No—Set 1. Yes—Crude soybean oil and diesel fuel, set 2 aerated for 48 hours; others not aerated.
USEPA/OPP 1982 (update 1985)	96 hours (96 hours)	(No, except aerate reconstituted water prior to use).
ASTM 1986	96 hours, except 48 hours for daphnids and midge larvae; record mortality at 24, 48, 96 hours for LC_{50}.	May gently aerate all chambers and controls; use simultaneous test without aeration; toxicant concentration in aerated chamber not more than 20% lower than unaerated.
USEPA/OTS 1985 (update 1987)	96 hours preferred, mortality at 24, 48, 72, 96 hours, LC_{50}, 95% confidence limits (96 hours).	Dilution water aerated until oxygen saturation, stored 2 days without further aeration.
USEPA/ORD 1985 (update 1991) {update 1993b}.	24–48 hours; 96 hours, some states (24–96 hours, depends on requirements).	May alter results, only as last resort; none, unless dissolved oxygen <4mg/l, at which time gentle single-bubble aeration (Aeration rate not over 100 bubbles/min in all test solutions).
APHA 1989	96 hours for LC_{50}; 24 hours, range-finding	Avoid aerating, because aeration may alter results.
OECD 1984	96 hours preferred; mortality recorded at 24, 48, 72, and 96 hours and LC_{50}.	May be used if no significant loss of test substance; must show test substance concentration at least 80% nominal concentration over test period.

Method	Static test duration	Aeration
EEC 1984	96 hours preferred, 48 hours minimum; morality recorded each 24 hours and $LC_{.50}$.	

Method	Test Vessels	Dissolved oxygen
AQUA Report 1993	Polyethylene buckets	Protocol says not below 4.5 mg/l (but was below 4.5 in 100% beef tallow and all concentrations of crude soybean oil, Set 1).
USEPA/OPP 1982 (update 1985)	(Glass or welded stainless steel; polyethylene absorbs test materials; for other materials, analyze toxicant concentration).	Measure concentration at start and every 48 hours to end; first 48 hrs., 60–100% saturation, then 40–100% (Measure in control, high, medium, low concentration).
ASTM 1986	Welded stainless steel or glass; size and shape of chamber may affect results if toxicant volatilizes or sorbs onto chamber.	60–100% saturation for first 48 hours, 40–100% saturation after 48 hours.
USEPA/OTS 1985 (update 1987)	Not contain substances that leached or dissolved into aqueous solutions or chemical sorption; glass, stainless steel, perfluorocarbon plastic.	Maintain above 4.5 mg/l or at least 60% air saturation value.
USEPA/ORD 1985 (update 1991) {update 1993b}.	Usually soft glass {Borosilicate glass or non-toxic disposable plastic, covered}.	4 mg/l minimum warmwater species, 6 mg/l minimum coldwater species.
APHA 1989	No material with leachable substances or adsorbs substances from water; stainless steel probably best, glass adsorbs organics; do not use rubber or plastics with fillers, additives, stabilizers..	At or near saturation, never below 4 mg/l or 60% saturation.
OECD 1984	Chemically inert materials, suitable capacity	At least 60% of air saturation value throughout.
EEC 1984		At least 60% of air saturation value at selected temperature throughout.

Method	Dilution Water	Chemical Analysis of Concentration
AQUA Report 1993	72 mg/l $CaCO_3$ (moderately hard, lab fresh water deionized).	None reported; nominal concentrations listed in report.
USEPA/OPP 1982 (update 1985)	Describe source, characteristics, pretreatment (Reconstituted water, soft, aged 1–2 weeks, aerated before use or natural water, hardness 40–48 mg/l as $CaCO_3$; animals not stressed).	Describe methods, concentration, validation and blanks if done (Chemical analysis of test solutions preferred, especially if aerated, material insoluble, containers not stainless steel or glass, or chemical adsorbs to container).
ASTM 1986	Test organisms survive without stress or grow and reproduce; reconstituted, surface, or natural water, requirements described.	Measure concentration at beginning and end in all chambers if possible; desirable to measure degradation products and report methods of analysis, standard deviation and validation studies.
USEPA/OTS 1985 (update 1987)	Drinking, natural, or reconstituted water, 50–250 mg/l as $CaCO_3$, pH6–8.5 preferred.	Measure concentration in each at beginning and end; validate analytical methods, degradation products not interfere; replicates within 20% (Concentration in each chamber not vary >30% from measured at start).
USEPA/ORD 1985 (update 1991) {update 1993b}.	Receiving water, other surface water, ground water, soft synthetic water {Same water, culturing and dilution}.	Use methods in CWA Sec 304(h) for analysis {Measure in each test concentration at start, daily, and end}.
APHA 1989	Reconstituted or natural water; standard water conditions for comparative toxicity, sensitivity tests.	Measure concentration in each container at start and once during test; measured concentration within 15% of calculated.
OECD 1984	Drinking, natural or reconstituted water; prefer hardness 50–250 mg $CaCO_3$ per liter, pH 6–8.5.	Must show concentration maintained and measured concentration at least 80% of nominal.
EEC 1984	Drinking water, natural water, reconstituted water; prefer 50–250 mg/l as $CaCO_3$, pH 6–8.5.	Evidence from analysis, chemical properties, or test system used that concentration maintained and within 80% of initial concentration.

Method	Results reported
AQUA Report 1993	48-hour LC_{50}; no confidence limits reported, but protocol says intervals computed.
USEPA/OPP 1982 (update 1985)	Effect criteria, percent with effects; 96-hour LC_{50}, 95% confidence limits, slope or show LC_{50}>100 mg/l (at least 30 organisms exposed) or >100,000 times maximum expected environmental concentration or estimated environmental concentration (Methods, materials, organisms, LC_{50}, 95% confidence limits, slope, calculations, chemical analysis).
ASTM 1986	24, 48, and 96-hour LC_{50}, 95% confidence limits, percentage died at each concentration and controls, calculation methods, and detailed information on test and organisms and findings, validation studies for analytical methods and accuracy.
USEPA/OTS 1985 (update 1987)	Test procedures and conditions, preparation of test solutions, maximum concentration with 0% mortality, minimum concentration with 100% mortality, cumulative mortality each concentration and time, LC_{50} based on nominal concentration at each time, 95% confidence limits, concentration-mortality curve at end, procedures for determining LC_{50}, mortality of controls, test according to guidelines.

Method	Results reported
USEPA/ORD 1985 (update 1991) {update 1993b}.	Chemical analysis, organisms died or effect in each chamber, observations, LC_{50}, 95% confidence intervals and methods to calculate, deviation from methods {Raw toxicity data, relationship between LC_{50} and NOAEL if NOAEL, pass/fail}.
APHA 1989	LC_{50}'s for exposure times, 95% confidence limits; mortality in controls, describe test conditions and methods, observations, test material, response criteria.
OECD 1984	Cumulative percent mortality vs. concentration; LC_{50}; confidence limits, p=0.95; where data inadequate, geometric mean of highest concentration with 0% mortality and lowest concentration with 100%.
EEC 1984	Methodology, highest concentration with 0% mortality, lowest concentration with 100% mortality, cumulative mortality, control, LC_{50}, 95% confidence limits, LC_{50} calculations, dose-response at end, slope, dissolved oxygen and pH and temperature every 24 hours.
Method	**Special considerations**
AQUA Report 1993	
USEPA/OPP 1982	Required to register end-use pesticide product introduced directly into aquatic environment, LC_{50} below or
(update 1985)	equal to maximum expected environmental concentration, or ingredient enhances toxicity
	(Required if insoluble; flow-through if high BOD; 17–22 °C, at least 10 organisms/concentration, loading limits; reviews statistical analysis; invalid if aerated or not glass or solubility problems).
ASTM 1986	Use flow-through if chemical has high BOD; loading limits specified so dissolved oxygen acceptable, metabolic products not above acceptable level, and no crowding; temperature not vary > 1°C; 10 organisms per concentration group.
USEPA/OTS, 1985	Guidelines for development of test rules standards, test data under Toxic Substances Control Act; loading
(update, 1987)	limits; 23° ± 2°C.
USEPA/ORD 1985	For National Pollutant Discharge Elimination System effluents; definitive vs. screening tests; loading, limits;
(update 1991)	20° C; 2 replicates, 10 organisms/concentration.
{update 1993b}	{If pH outside 6–9, two parallel tests, one adjusted; or static renewal or flow-through}.
APHA 1989	5 concentrations and control; 10 fish/tank, 20 fish/concentration; species in receiving water or similar, available for tests, healthy in lab, important trophic link or economic resource.
OECD 1984	21–25° C; carry out without pH adjustment, adjust pH of stock solution if necessary so concentration not changed and no reaction or precipitation.
EEC 1984	20–24 °C ± 1°C; carry out without pH adjustment, adjust if necessary; interpret results with care if stability or homogeneity of test substance not maintained.

[1] In some instances, other test conditions were allowed (USEPA, 1996). Draft Amendment to Standard Evaluation Procedures, 1996 states: Individual fish should weigh 0.1–5 g. Hardness of natural dilution water of less than 200 mg/l as CaCO3 can be used in lieu of reconstituted water for organic chemicals. Chemicals that are poorly soluble or with a water solubility less than 100 ppm (<100 mg/l) should be tested up to the maximum water solubility if certain conditions apply.

[2] Final Report of Fourth Edition, August, 1993.

TABLE 4.—EFFECTS OF REAL-WORLD OIL SPILLS

Name and location of spill	Oil spilled	Effects
Minnesota Soybean Oil and Petroleum Oil Spills (1962–1963).[1,2]	1 to 1.5 million gallons soybean oil from storage facilities, 1 million gallons low viscosity cutting oil.	Killed thousands of ducks and other waterfowl and wildlife or injured them through coating; 5,300 birds injured or died, 26 beavers, 177 muskrats.
		Formed stringy, rubbery masses with slicks; sank to bottom; milky material and hard crusts of soybean oil with sand on beaches.
		Soybean oil caused much of waterfowl loss, as shown by lab analysis of oil scraped from ducks.
Fanning Atoll Spill (1975).[3]	Cargo ship with coconut oil, palm oil, and edible materials; ran aground, dumped cargo onto coral reef.	Effects similar to petroleum oil spill.
		Killed fish, crustaceans, mollusks; shifts in algal community continued for 11 months.
Kimya Spill, North Wales (1991).[4,5,6,7,8]	Cargo of unrefined sunflower oil ...	Killed mussels, shifts in ecological communities around spill.
		Polymerized, covered bottom, killed benthic organisms; formed impermeable cap, shut out oxygen, bacteria cannot break down; polymers remain nearly 6 years later.
		Concrete-like aggregates of oil and sand on beach.
		Lab studies of mussels show small amounts of sunflower and other vegetable oils kill mussels after 2 weeks; affect mussel lining.
Rapeseed Oil Spills (1974–1978).[9]	3 small spills, total about 35 barrels rapeseed oil.	Greater losses of birds from 3 small spills of rapeseed oil than 176 spills of petroleum oils over 5 years in Vancouver Harbor.
		Killed 500 birds; petroleum spills killed less than 50 birds.
		Perhaps vegetable oils lack strong, irritating odor of petroleum oils, so birds do not avoid.
(1989).[10]	About 10 barrels (400 gallons) of rapeseed oil.	88 oiled birds of 14 species, half of them dead; half of rescued birds died; casualties probably higher.
		About 300 oiled Barrow's Goldeneyes spotted 2 days after spill crowded onto islands where they remained for 2 days—fate unknown, but weakened birds often die.

TABLE 4.—EFFECTS OF REAL-WORLD OIL SPILLS—Continued

Name and location of spill	Oil spilled	Effects
Fat and Oil Pollution in New York State Waters (1967).[11]	Wide variety of sources.	Killed waterfowl, coated boats and beaches, tainted fish, created taste and odor problems in water treatment plants. Grease like substances on shore or floating on Lake Ontario; shoreline grease balls smelled like lard, analyzed as mixtures of animal and vegetable fats.
Spills of Fish Oil Mixtures near Bird Island, Lamberts Bay, South Africa (1974).[12]	Fish factory effluent pipe near breeding ground for Cape Gannets.	Killed at least 709 Cape Gannets, 5,000 Cape Cormorants, and 108 Jackass Penguins. Penguins with sticky, white, foul-smelling coat of oil shivering; gannet chicks dead. Milky white sea and clots of oil on island smelling of fish.
Releases at two other fish factories at St. Helena Bay and Saldanha Bay, South Africa (1973).[13]	Two other fish factories; storage pits and processing effluents and off loading water from vessels.	Two other fish factories; at one, killed 10,000 rock lobsters and thousands of sea urchins probably from oxygen depletion; at second, killed 100,000 clams and black mussels, prawns, polychetes, and anemones, and smelled bad and adversely affected aesthetics of beaches and camping site.
Soybean Oil Spills in Georgia (1996).[14]	Soybean oil from tanker truck and soybean vegetable oil refinery with overfilled aboveground storage tank.	Aesthetic effects at Lake Lanier; rancid oil as weathered; adhered to boats and docks. At Macon, rapid response prevented significant damage from oil, which flowed through storm water system and entered stream; previous spills from facility had entered sanitary sewer system and damaged sewage treatment plant.
Spill of Nonylphenol and Vegetable Oils in Netherlands (December,1988 to March, 1989).[15]	Unknown source	Thousands of seabirds, mostly Guillemots and Razorbills, washed ashore. 1,500 sick birds died; covered with oil, emaciation, aggressive behavior, bloody stools, leaky plumage; liver damage, lung infections. High levels of nonylphenol and vegetable oils, such as palm oil.
Wisconsin Butter Fire and Spill (1991).[16,17,18,19,20,21,22,23]	Butter, lard, cheese as well as meat and other food products.	Released 15 million pounds of butter and 125,000 pounds of cheese into the environment and damaged at least 4.5 million pounds of meat; thousands of pounds of butter ran offsite; rapid response prevented flow of buttery material through storm sewers to nearby creek and lake, where fish and other aquatic organisms could have suffocated from oxygen depletion. Destroyed two large refrigerated warehouses with $10 million to $15 million in property damage. Cost tax payers $13 million for butter and cheese stored under USDA surplus program. Damage to fire equipment from grease, loss of business, overtime pay for 300 firefighters and responders, costs for cleaning equipment and drains, rodent control. Environmental cleanup costs; thousands of gallons of melted butter; butter and spoiled meat declared hazardous waste.

[1] Minnesota, 1963.
[2] USDHHS, 1963.
[3] Russell and Carlson, 1978.
[4] Salgado, 1992.
[5] Mudge et al., 1993.
[6] Mudge et al., 1995.
[7] Mudge, 1997a.
[8] Mudge, 1997b.
[9] McKelvey et al., 1980.
[10] Smith and Herunter, 1989.
[11] Crump-Wiesner and Jennings, 1975.
[12] Percy-Fitzpatrick Institute, 1974.
[13] Newman and Pollock, 1973.
[14] Rigger, 1997.
[15] Zoun et al., 1991.
[16] Wisconsin, 1991a.
[17] Wisconsin, 1991b.
[18] Wisconsin, 1991c.
[19] Wisconsin State Journal, 1991a.
[20] Wisconsin State Journal, 1991b.
[21] Wisconsin State Journal, 1991c.
[22] Wisconsin State Journal, 1991d.
[23] Wisconsin State Journal, 1991.

Appendix II—Edible Oil Regulatory Reform Act Differentiation

Edible Oil Regulatory Reform Act

Congress enacted the Edible Oil Regulatory Reform Act on November 20, 1995. The Act requires all Federal agencies (with the exception of the Food and Drug Administration) to (1) differentiate between and establish separate classes for animal fats and oils and greases, fish and marine mammal oils, oils of vegetable origin, including oils from certain seeds, nuts, and kernels, from other oils and greases, including petroleum; and (2) apply standards to different classes of fats and oils based on certain considerations. In

differentiating between the classes of fats, oils, and greases, each Federal agency shall consider differences in the physical, chemical, biological, and other properties, and in the environmental effects, of the classes. These requirements apply when Federal agencies are issuing or enforcing any regulation or establishing any interpretation or guideline relating to the transportation, storage, discharge, release, emission, or disposal of a fat, oil, or grease under any Federal law.

EPA's Final Rule amending the Oil Pollution Prevention regulation (Oil Pollution Prevention; Non-Transportation-Related Onshore Facilities; Final Rule, 59 FR 34070, July 1, 1994) was promulgated before the Edible Oil Regulatory Reform Act was enacted; Congress did not make the requirements of the Act retroactive. EPA is, therefore, not obligated to evaluate the statutory criteria to determine if a further differentiation between edible oils and other oils should be made in its Final Rule. EPA does, however, present the following information in support of its conclusion that spills of vegetable oils and animal fats can indeed pose a serious risk to fish, wildlife, and sensitive environments.

A summary of the properties and effects of vegetable oil and animal fats are presented in Appendix I, Tables 1 and 2. Additional detailed discussion and studies of these properties and effects are contained in the Technical Document in support of this document.

Physical Properties. Vegetable oils and animal fats are generally solids in water at ambient temperatures. They both have limited water solubility but high solubility in organic solvents. They generally are of low viscosity, have a low evaporation potential, and their specific gravity can range from 0.87 to 0.92. Petroleum oils also have limited water solubility and high solubility in organic solvents. They form an emulsion in turbulent water, and they evaporate faster than edible oils. Their specific gravity can range from 0.78 to 0.97. Data regarding petroleum oil's solidity and viscosity vary. (See Appendix I, Table 1. Comparison of Physical Properties of Vegetable Oils and Animal Fats with Petroleum Oils and Table 2. Comparison of Vegetable Oils and Animal Fats with Petroleum Oils.

Vegetable oils and animal fats and petroleum oils all have similar physical properties. One difference is the low volatility of most vegetable oils and animal fats, which results in less product removed from a spill by evaporation and reduces the combustion and explosive potential of these oils.

Chemical Properties. Animal fats and vegetable oils are water-insoluble substances that consist predominantly of glyceryl esters of fatty acids or triglycerides. Petroleum oils are extremely complex mixtures of chemical compounds. Many classes of compounds are present in petroleum, and each class is represented by many components. For example, hydrocarbons are a major class of constituents of petroleum. Similar behavior of fatty acids and petroleum oil in the aquatic environment is largely a result of their predominantly hydrocarbon character.

Biological Properties. Some vegetable oils and animal fats do biodegrade more readily than petroleum oils; however, because their evaporation potential is low, vegetable oils and animal fats may tend to stay in the water in larger quantities and for longer periods of time than petroleum oils. Under certain circumstances, vegetable oils and animal fats can remain in the environment for periods of time greatly exceeding their potential degradation time. Environmental circumstances play an important part with regard to the comparative degradation rates of petroleum and non-petroleum oils including vegetable oil and animal fats. Both kinds of oil degrade more slowly in low-energy and poorly oxygenated waters, and both tend to disappear quickly in high-energy, well oxygenated, open water areas. Both petroleum and non-petroleum oils can remain in the environment for extended periods of time if buried under sediment or spilled in large enough quantities to form thick layers. The high BOD of vegetable oils and animal fats increases the rate of biodegradation but also quickly depletes the available oxygen of the surrounding environment. This could result in significant harm to shallow near-shore areas or wetlands. Oxygen depletion could be as serious as toxicity with regard to its impact on aquatic wildlife.

Environmental Effects. Certain effects of non-petroleum oils are similar to the effects of petroleum oils because of the physical properties common to both. Significant environmental harm from petroleum oils, animal fats and vegetable oils, and other non-petroleum oils can occur as a result of the following: physical effects such as coating with oil, suffocation, contamination of eggs and destruction of food and habitat, short and long term toxic effects, pollution and shut down of drinking water supplies, rancid smells, fouling of beaches and recreational areas.

Summary of Analysis after Reviewing the Act's Criteria. Based on the significant degree of similarity between animal fats and vegetable oils and other petroleum and non-petroleum oils, especially with respect to negative environmental effects associated with the common physical properties of all oils, EPA stands by its decision not to make further changes to its July 1, 1994, Final Rule. The Final Rule already provides a greater degree of flexibility for owners or operators of facilities storing only non-petroleum oils, including vegetable oils and animal fats, to devise different and more appropriate response strategies than owners or operators of petroleum oil facilities.

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